

EVALUATION OF COMA IN NON HEAD INJURY CASES

Dissertation submitted to
The Tamilnadu Dr.M.G.R. Medical University

In partial fulfillment of the regulations

For the award of the degree of

M.D. General Medicine – [Branch- 1]

DEPARTMENT OF GENERAL MEDICINE

K.A.P.VISWANATHAM GOVERNMENT MEDICAL COLLEGE

& M.G.M.GOVERNMENT HOSPITAL,

TIRUCHIRAPALLI



**THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY
CHENNAI**

APRIL – 2016

BONAFIDE CERTIFICATE

Certified that the dissertation titled “**EVALUATION OF COMA IN NON HEAD INJURY CASES**” is a bonafide work done by **Dr SREETHA SREENIVAS**, under my guidance and supervision, in partial fulfilment of regulations of The Tamil Nadu Dr MGR Medical University for the award of M D Degree Branch 1(General Medicine) during the academic period 2013-2016

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DECLARATION

I solemnly declare that the dissertation titled “**EVALUATION OF COMA IN NON HEAD INJURY CASES**” was done by me at K A P V Government Medical College attached to MGM Govt.Hospital,Tiruchirappalli under the guidance and supervision of **Prof.Dr K.PARIMALADEVI MD.** The dissertation is submitted to The Tamil Nadu Dr M G R Medical University towards partial fulfilment of the requirement for the award of M D Degree In General Medicine

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ACKNOWLEDGEMENT

I sincerely thank **Prof. Dr.M K MURALIDHARAN MS MCh** (Neuro Surgery), **Dean**, K A P V Government Medical College ,Tiruchirappalli for having permitted me to undertake the study in this prestigious institution.

It is a great pleasure to express my sincere thanks to **Prof.Dr P KANAGARAJ MD**, Head of the Department of Medicine for his guidance and encouragement.

With extreme gratitude and respect, I express my indebtedness to my beloved Unit Chief, **Prof. Dr. K.PARIMALADEVI M D** for her motivation, guidance, and critical suggestions which enabled me to complete this work.

I am thankful to our medicine chiefs; **Prof.Dr G ANITHA MD**, **Prof.Dr.V.RAJENDRAN MD (Rtd)**, **Dr. N.K. SENTHILNATHAN MD**, **Dr.A.SETHURAMAN MD**, **Dr. D. NEHRU MD** for their valuable guidance and motivation.

I am thankful to **Dr. M. RAJASEKARAN MD DM** (Neurology) Registrar, Department of Medicine for his valuable guidance and encouragement.

I whole heartedly thank **Prof.Dr. S. ANGURAJ MD, DM** (Neurology) Head of Department ,Neurology and **Dr. P.V.KRISHNAN MD DM** (Neurology) and **Dr. E. ARUNRAJ MD DM** (Neurology) for their valuable support and suggestions.

I would like to express my gratitude to Asst. Professors **Dr.U.B.PADMANABAN MD,** **Dr.A.THENMOZHI MD,** **Dr. S. PALINIVELRAJAN MD** of my unit for their constant encouragement ,timely help and valuable criticism.

I sincerely thank the Department of Biochemistry and Department of Radiology for their cooperation and support. I thank my post graduate colleagues and CRRI'S for their help and suggestions.

I thank all patients who participated in this study and their relatives for their sincere cooperation

Last but not the least, I thank God.

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INTRODUCTION

INTRODUCTION

Human brain is a complex organ performing a multitude of functions responsible for maintaining the milieu interior and exterior. These functions are maintained both in the conscious and unconscious state. Consciousness requires an intact brain functions, or to be more precise, an intact ascending reticular activating system (ARAS). Any damage to this causes absent arousal or coma.

Patients can sometimes present with episodic impairment of consciousness. It can be due to

- A. Impaired cerebral perfusion-syncope
- B. Cerebral Ischemia
- C. Epilepsy
- D. Migraine
- E. Sleep disorders
- F. Suddenly increased ICT
- G. Psychogenic

Various cardiac, non cardiac or undetermined causes can result in syncope. A simple ECG can identify the multitude of cardiac causes resulting in syncope.

Seizures can also cause alterations in consciousness levels and this can be differentiated from syncope with history and examinations.

Migraine, Sleep disturbances, any cause of sudden increase in intracranial tension and psychogenic factors also cause transient loss of

consciousness. As with any other cause of loss of consciousness, patients with episodic loss of impaired consciousness also is evaluated from history by a reliable attender, biochemical and imaging modalities.

Comatose patient presenting to an emergency room is always a challenge to the attending physician, especially the non traumatic coma. Prompt and precise diagnosis helps to give adequate and appropriate treatment immediately, thereby increasing patients chance of recovery.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

- 1) To find out usual and rare causes of coma
- 2) To assess short term prognosis
- 3) To find out clinical tools which will add on to/increase the diagnostic yield

REVIEW OF LITERATURE

REVIEW OF LITERATURE

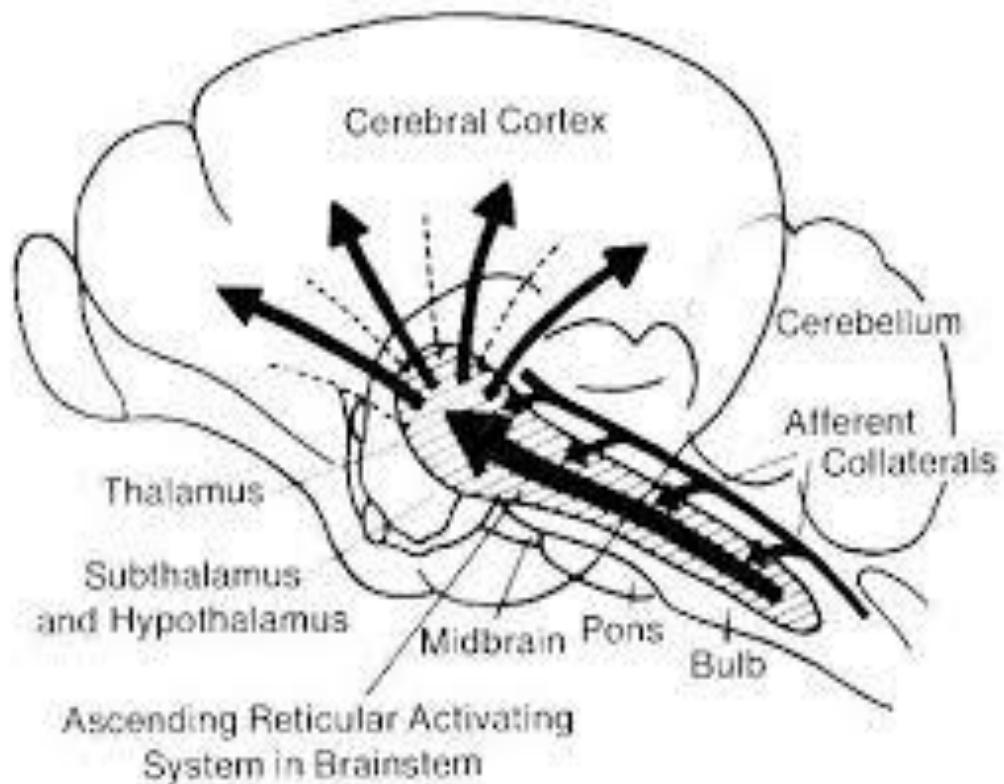
Consciousness is a state of awareness of one's own self and surroundings. It requires an intact and functioning brainstem ascending reticular activating system (ARAS) and its projections. Persistent level of decreased or absent consciousness shows disorder of the brainstem and diencephalon arousal mechanism and is called coma, if there is complete unresponsiveness to arousal.¹

Stupor is a higher degree of unarousability in which patient can be transiently awakened by vigorous stimuli.

Drowsiness is easy arousal and persistence of alertness for brief periods.

ANATOMY AND PHYSIOLOGY

Maintenance of consciousness depends on integrity and interaction between ARAS and cerebral hemispheres. In humans, ARAS lies in paramedian tegmental region of posterior part of midbrain and pons. Median longitudinal fasciculus which connect opposite third and sixth cranial nerve nuclei and third and fourth cranial nerve nuclei themselves are situated amidst the neurons of ARAS. So when brainstem damage cause coma, lesion affect ocular movement also.^{9, 10}



SCHEMATIC REPRESENTATION OF THE ASCENDING RETICULAR ACTIVATING SYSTEM

Based on anatomic structures and locations, coma can be due to: ²,

1. Lesions damaging ARAS in upper midbrain or its projections
2. Bi hemispherical damage, usually large portions.
3. Drugs/ toxins/ metabolic abnormalities that suppress reticular cerebral function.

CORRELATION BETWEEN CLINICAL SIGNS AND INVESTIGATIONS

SIGN	CSF FINDING	IMAGING
No local or lateralising neurological signs, Normal brainstem functions	normal	normal
Signs of meningeal irritation, usually without focal or Lateralising cerebral or Brainstem signs	excess WBC or RBC	may not show Mass lesion
Focal brainstem or lateralising Cerebral or brainstem signs	with or without changes	abnormal

MASS LESIONS

- Cause lateral displacement of midbrain → compresses contralateral cerebral peduncles → Babinski sign and hemiparesis opposite to original hemiparesis.
- Kernohan – Woltman sign.
- In acutely appearing masses – horizontal displacement of pineal body occurs.
- If mass is 3-5 mm → drowsiness
 6-8mm → stupor
 >9mm → coma

METABOLIC

- Coma occurs due to interruption of energy delivering substrates or altering excitability of neurons.⁵
- Neuropathological changes are usually minor and reversible.
- Many systemic medical disorders can result in large shifts in water and sodium balance in brain, resulting in seizures and coma.
- In metabolic encephalopathy the degree of neurological change depends largely on the rapidity with which changes occur in the serum.
- Pathophysiology of many causes of metabolic coma is largely not understood and may reflect derangements in biochemistry, neurotransmitters, neuromodulation and cell membrane functions.^{4,6}

Diagnosis of metabolic encephalopathy as a cause of coma means there has been no focal features to label an anatomical structure for the cause but also a specific metabolic cause can't be established. Such a state occurs with endogenous toxins.

Exogenous toxins have predilection for ARAS and reflex eye movements will be paralyzed.

SEIZURES

Coma can also happen following seizures – the so called post ictal state.

TOXIC OR DRUG INDUCED

Largely reversible and leaves no residual damage, provided there has been no cardiorespiratory failure.

CEREBRAL HEMISPHERICAL LESIONS: ⁸

Number of unrelated disorders cause widespread structural damage to cerebral hemispheres stimulating metabolic causes of coma.

Small vessel occlusion can also cause coma.

A few causes are:

- 1) Hypoxia- ischemia
- 2) Cerebral malaria
- 3) TTP
- 4) Hyper viscosity due to any cause

Inflammatory demyelinating diseases can cause diffuse white matter damage.

INFECTIONS / HAEMORRHAGE

Meningeal irritation caused by infection or hemorrhage is potentially treatable and it should be among the most important early consideration because they may not be diagnosed with imaging

CARDIAC ARREST

Coma can also occur following cardiac arrest and prognosis for a meaningful recovery can be assessed from clinical signs. In cardiac arrest patient who don't have seizures, return of pupillary reactivity and purposeful motor movements within first 72 hours correlate with good outcome.

CVA

Diseases that cause coma are:

1. Basal ganglia and thalamic hemorrhage
2. Pontine hemorrhage
3. Cerebellar hemorrhage
4. Basilar artery thrombosis
5. SAH
6. Massive infarct with cerebral edema
7. Massive infarct/hemorrhagic infarct with midline shift
8. Any massive ICH with intraventricular extension

HISTORY AND EXAMINATION

History is obtained from an attender or a person who witnessed the event as the case with seizures.

Preceding headache, confusion or delirium points towards meningitis or toxins.

Sudden cause of coma is suggestive of stroke affecting brainstem, subarachnoid hemorrhage, and intracerebral hemorrhage with intraventricular extension.^{12, 13}

Localizing features like aphasia can occur with hemispheric masses.

Signs of meningeal irritation if able to elicit will point towards important clues to diagnosis like meningitis and SAH.

Mass lesions which are expanding will show progressive clinical features which helps in identifying the anatomic structure which is being compressed. ;eg:pituitary tumor compressing optic chiasma.

Metabolic causes won't usually result in progressive clinical features and they rarely produce asymmetric motor signs similar to mass lesions.

Investigations like CT and MRI brain, EEG will help in reaching a conclusion about diagnosis ^{7, 11}

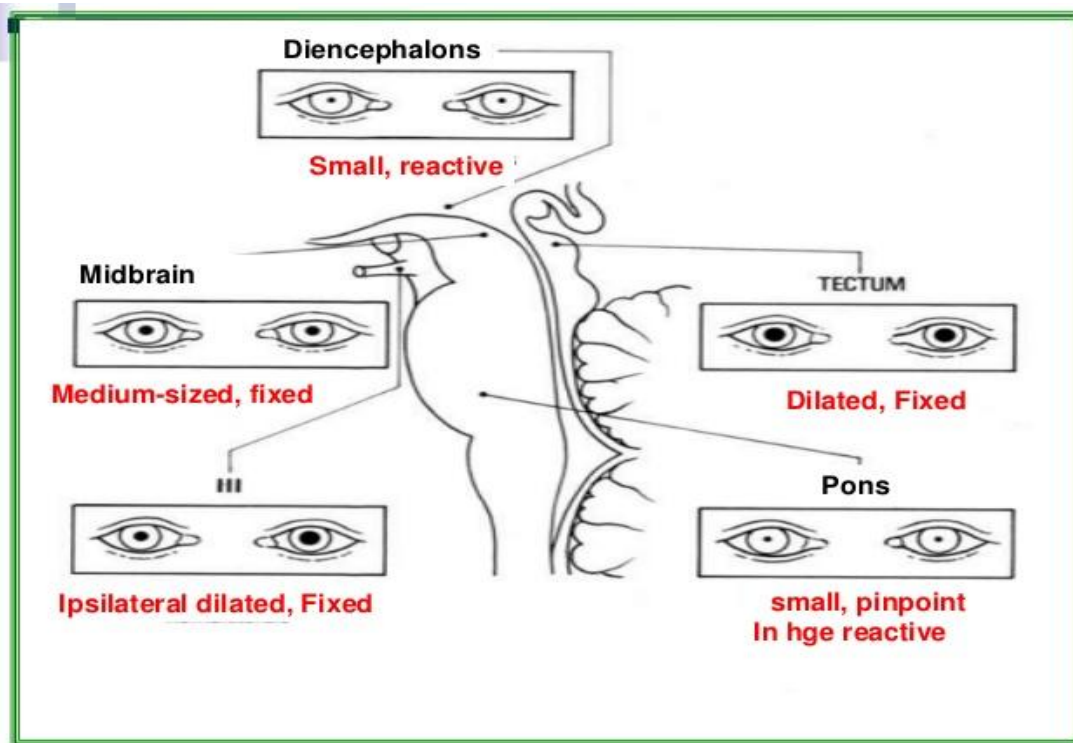
PUPILS

In metabolic coma early stage, pupillary reactivity is retained and is lost only when coma is very deep in which patient requires ventilatory and inotropic support.

Functioning pupil means intact midbrain.

PUPILLARY ABNORMALITIES AT DIFFERENT LEVELS: ¹⁵

1. Small, reactive - diencephalic/metabolic
2. Midposition, unreactive, spontaneous hippus-midbrain tectum
3. Midposition, irregular, unreactive, -midbrain tegmentum
4. Ipsilateral large unresponsive - fascicular/peripheral third nerve
5. Pinpoint, responsive - pons



PUPILLARY ABNORMALITIES WITH LESIONS AT VARIOUS LEVELS

SPONTANEOUS EYE MOVEMENTS:^{19,20,21,22}

If purposeful eye movements are present in an otherwise unresponsive patient, coma mimics should be considered in the diagnosis.

Roving eye movements are slow lateral conjugate to and fro excursions. These occur when third nerve nuclei and connections are intact and often indicate a toxic, metabolic or alternatively bilateral hemispherical cause of coma.

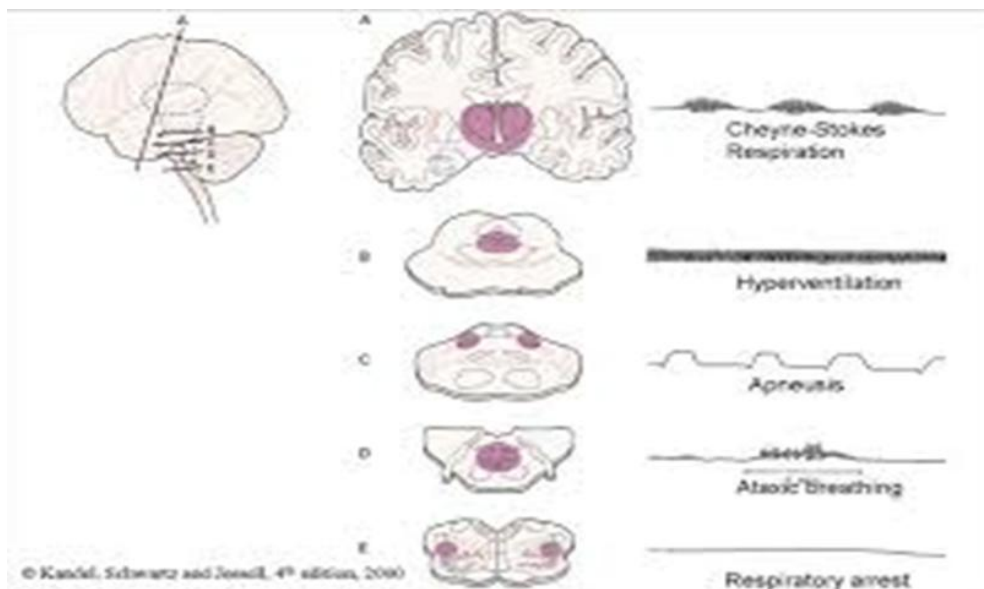
Ocular bobbing describes a rapid downward jerk of both eyes with slow return to the mid position. this is specific for acute pontine lesions.

REFLEX EYE MOVEMENTS: ¹⁵

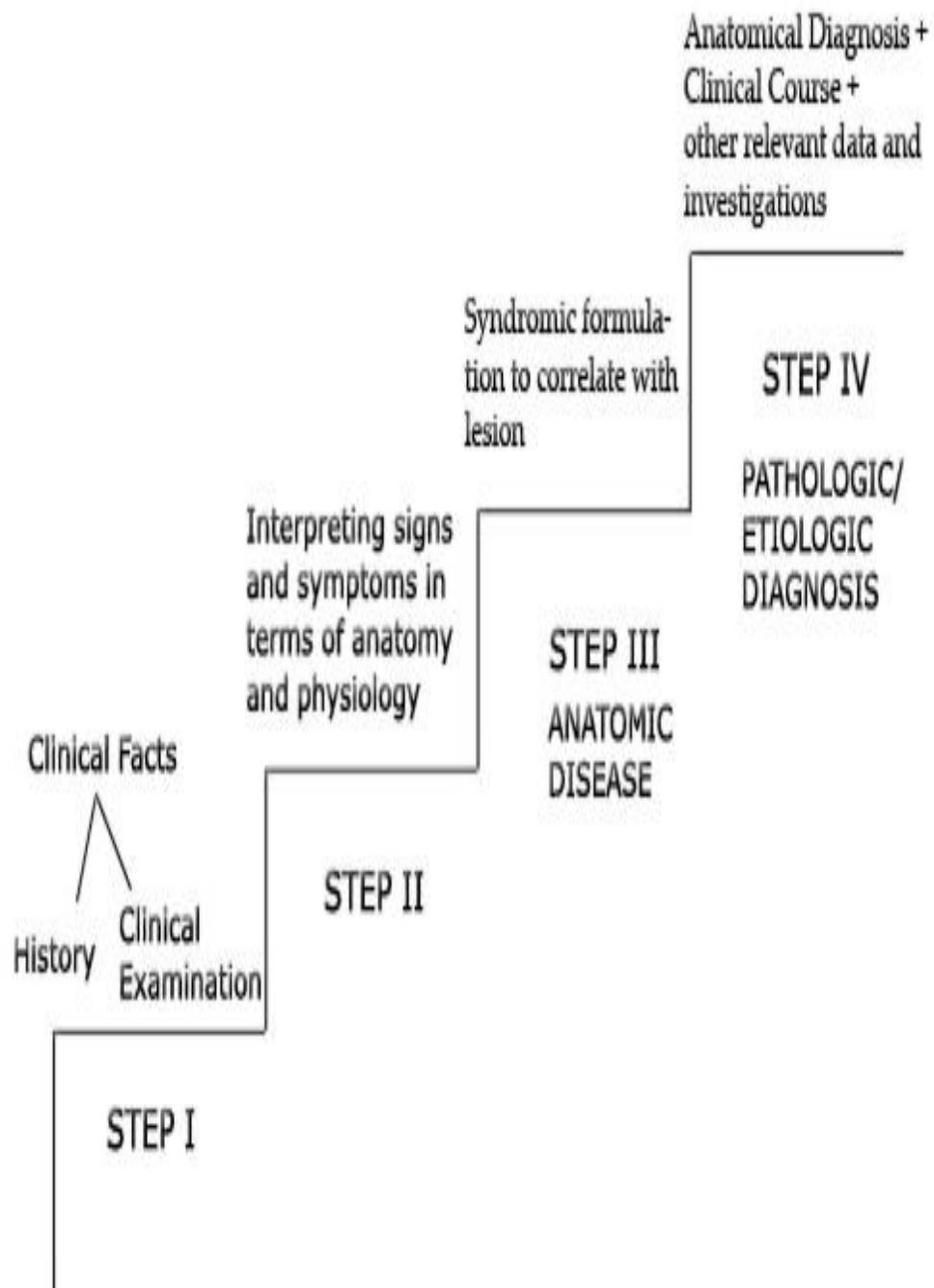
- 1) Dolls eye movements reflect integrity of pons.
- 2) In metabolic coma, reflex eye movements can be lost or retained.

ABNORMAL RESPIRATORY PATTERNS: ¹⁴

1. Cheyne-Stokes respiration: diffuse forebrain damage.
2. Central neurogenic hyperventilation: from midbrain ventral to aqueduct of Sylvius and of upper pons, ventral to fourth ventricle.
3. Apneustic: dorsolateral tegmental lesion of middle and caudal pons.
4. Cluster breathing: lower pontine tegmental lesion.
5. Ataxic breathing: reticular formation of dorsomedial part of medulla



STEPS IN DIAGNOSIS OF NEUROLOGIC DISEASES



CAUSES OF COMA

1)SYMMETRICAL STRUCTURAL

A)TOXINS

- Lead
- Thallim
- Mushrooms
- Ethanol
- Cyanide
- Methanol
- Ethylene glycol
- Carbon monoxide

B)DRUGS

- Sedatives
- Barbiturates
- Other hypnotics
- Tranquillizers
- Alcohol
- Opiates
- Paraldehyde
- Anticholinergics
- Psychotropes
- Amphetamines

- Lithium
- Phencyclidine
- Monoamine oxidase inhibitors

C)METABOLIC

- Hypoxia
- Hypercapnia
- Hyponatremia
- Hyponatremia
- Hypoglycemia
- Hyperglycemic nonketotic coma
- Diabetic ketoacidosis
- Hypercalcemia
- Hypocalcemia
- Hypomagnesemia
- Hyperthermia
- Hypothermia
- Hepatic encephalopathy
- Reyes encephalopathy
- Wernickes encephalopathy
- Aminoacidemia
- Porphyria
- Dialysis encephalopathy
- Addisonian crisis

D)INFECTIONS

- Meningitis
- Encephalitis
- Post infectious encephalomyelitis
- Syphilis
- Malaria
- Typhoid
- Sepsis
- Waterhouse frederichson syndrome

E)PSYCHIATRIC

- Catatonia

F)OTHERS

- Post ictal
- Diffuse ischemia
- Hypotension
- Fat embolism
- Hypothyroidism
- Hypertensive encephalopathy

2)SYMMETRICAL NON STRUCTURAL

A)SUPRATENTORIAL

- Bilateral internal carotid artery occlusion
- Bilateral anterior cerebral artery occlusion

B)INFRATENTORIAL

- Basilar occlusion
- Pontine hemorrhage
- Midline brainstem tumor

C)SUBARACHNOID HEMORRHAGE

- Thalamic hemorrhage
- Trauma-concussion,contusion
- Hydrocephalus

3)ASYMMETRICAL STRUCTURAL

A)SUPRATENTORIAL

- Thrombotic thrombocytopenic purpura
- Disseminated intravascular coagulation
- Non bacterial thrombotic endocarditis(Marantic endocarditis)
- Sub acute bacterial endocarditis
- Fat emboli
- Unilateral hemispheric mass with herniation

B)SUB DURAL EMPYEMA

- Brainstem infarction
- Brainstem hemorrhage
- Multiple sclerosis
- Thrombophlebitis
- ADEM
- Leukoencephalopathy associated with chemotherapy

C)SUB DURAL HEMORRHAGE-BILATERAL

- Intra cerebral bleed
- Pituitary apoplexy
- CJD
- Cerebral abscess
- Cerebral vasculitis
- Adrenal leucodystrophy
- Massive or bilateral supratentorial infarction

COMA MIMICS

Sl. No.	COMA MIMICS	LESION	FEATURES
1	Locked in syndrome	Below reticular formation	Voluntary eye movements preserved, especially vertical movements
2	Psychogenic	-	Reactive pupils
3	Persistent vegetative state	ARAS intact but cortical connections interrupted	Eye opening present. Brainstem reflexes intact
4	Akinetic mutism	Medial thalamic nucleus/ frontal lobe	Able to form impressions and think but virtually immobile and mute
5	Abulia	Frontal lobe and its connections	Diminished ability to initiate activity
6	Catatonia	-	Waxy flexibility of limbs

FOUR SCORE COMA ASSESMENT SCALE [FOUR-Full Outline of UnResponsiveness]

RESPONSE

E₄: eyes opened or unopen, tracking or blinking to command

E₃: eyelids open but not tracking

E₂: eyelids close but open to pain

E₁: eyelids remain closed with pain

MOTOR RESPONSE

M₄: thumbs up fist or peace sign

M₃: localizing to pain

M₂: flexion to pain

M₁: extension to pain

M₀: no response to pain or generalized myoclonic status epilepticus

BRAINSTEM REFLEXES

B₄: pupillary and corneal reflexes present

B₃: one pupil dilated and unreactive to light

B₂: pupillary or corneal reflex absent

B₁: pupillary and corneal reflex absent

B₀: absent pupillary, corneal and cough reflexes

RESPIRATION:

R₄: regular breathing pattern

R₃: Chyne – Stokes breathing pattern

R₂: irregular breathing pattern

R_1 : triggers or breathes above ventilator rate

R_0 : apnea or breathes at ventilator rate

(For non-traumatic coma and other disorders of consciousness)

INTERPRETATION

The lower the score, in each, poorer the prognosis.

GLASGOW COMA SCALE

BEST EYE RESPONSE

4 – Opens spontaneously

3 – to verbal commands

2 – to pain

1 – No eye opening

BEST VERBAL RESPONSE

5 – Oriented

4 – Confused

3 – Inappropriate words

2 – Incomprehensible sounds

1 – None

BEST MOTOR RESPONSE

6 – Obeys commands

5 – Localizes pain

4 – Withdraw from pain

3 – Abnormal flexion

2 – Abnormal extension

1 – None

[For trauma due to head injury]

INTERPRETATION:

Maximum score: 15

Minimum score: 3

Comatose: scores <8

AVPU SCALE

A-alert ;corresponds to GCS of 15

V-voice response;corresponds to GCS 12

P-pain response;corresponds to GCS 8

U-unconsciousness;corresponds to GCS 3

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA.

This study was conducted at Mahatma Gandhi Memorial Govt.Hospital attached to K A P V Govt.Medical College Trichy

STUDY DESIGN

Descriptive study

PERIOD OF STUDY

November 2014 to August 2015

ETHICAL COMMITTEE APPROVAL

Approval was obtained from Institutional Ethics committee

INCLUSION CRITERIA

Age >12 yrs <80 yrs

Comatose patients presenting to emergency room with non traumatic causes.

Consent for the study

EXCLUSION CRITERIA

Age<12 yrs

Coma due to head injury

CONSENT

An informed consent was obtained from relatives of all participants

METHOD

In this study, 60 participants aged >12 years presented to emergency room in Medicine from November 2014 to August 2015 were evaluated after getting informed consent from legal guardian. History taking and clinical examination was done and recorded in the form of proforma. History included age, sex, duration of illness/duration since last seen as normal, precipitating factors or events, symptoms preceding illness, risk factors. Detailed head to foot examination, Neurological and other system examination was done. Consciousness level was assessed using GCS, AVPU Scale and FOUR score coma assessment scale at admission, 24, 48 and 72 hours depending on individual patients and prognosis evaluated. CT scan brain was done for all cases at admission to find out any structural causes and blood investigations to elucidate non structural causes of coma.

STATISTICAL ANALYSIS

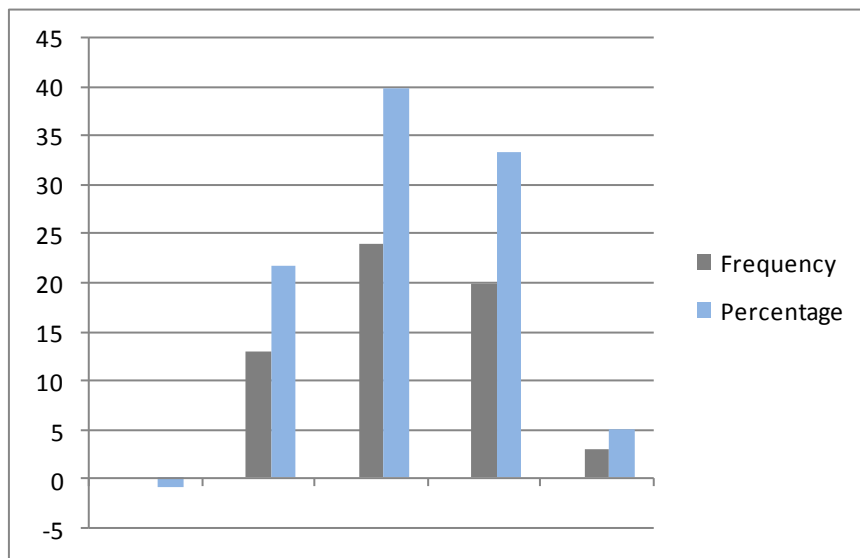
Statistical analysis was done using percentage, mean values, standard deviation, standard error, Chi square test. SPSS version 2 was used to analyse data. The level of significance used was 0.05 levels for the corresponding degree of freedom to draw inference. A p value < 0.05 was considered significant statistically and value > 0.05 was considered to be not statistically significant.

OBSERVATION AND INFERENCES

OBSERVATION AND INFERENCES

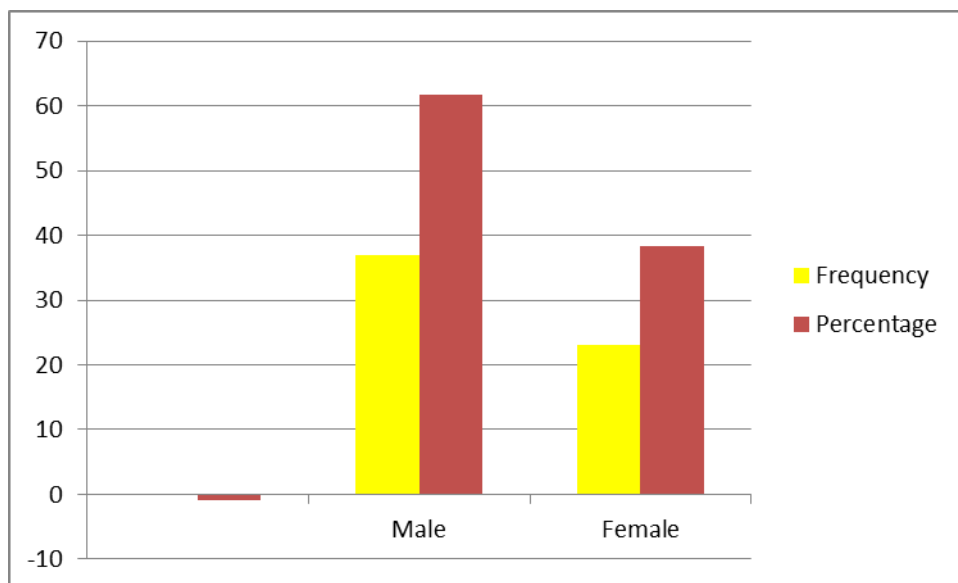
AGE (1a)

Particulars	Frequency (n=60)	Percentage -100%
13 to 33	13	21.7
34 to 54	24	40
55 to 75	20	33.3
76 & above	3	5



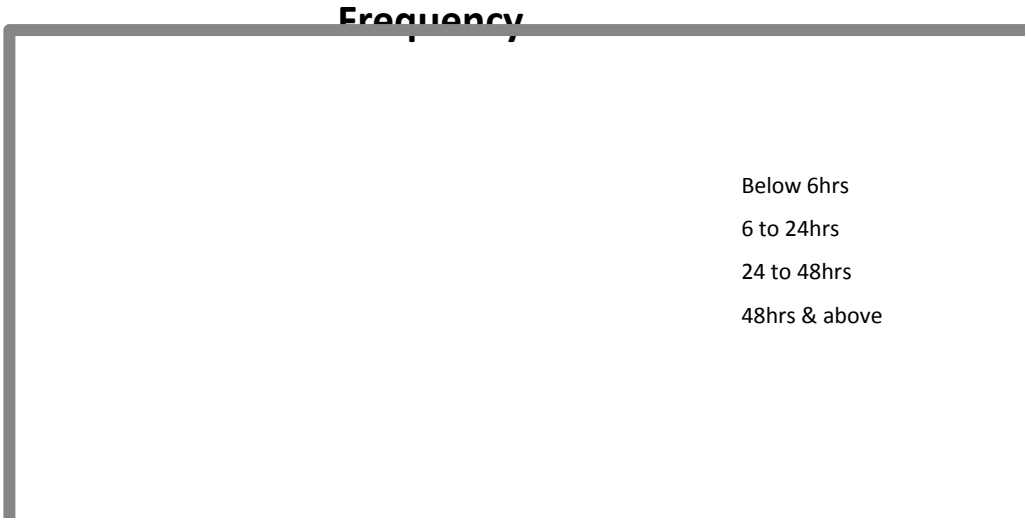
SEX(1b)

Particulars	Frequency (n=60)	Percentage -100%
Male	37	61.7
Female	23	38.3



DURATION OF ILLNESS(1c)

Particulars	Frequency (n=60)	Percentage -100%
Below 6hrs	27	45
6 to 24hrs	11	18.3
24 to 48hrs	11	18.3
48hrs & above	11	18.3



SYMPTOMS(1d)

Particulars	Frequency (n=60)	Percentage -100%
BREATHLESSNESS	8	13.3
FEVER	3	5
FEVER HEADACHE	5	8.3
FEVER,BODY STIFFNESS	2	3.3
FEVER,DYSURIA	1	1.7
FEVER,HIGH COLORED URINE	1	1.7
INCREASED SALIVATION	1	1.7
INCREASED SWEATING,SALIVATION	2	3.3
JAUNDICE	1	1.7
NO SYMPTOMS	24	40
TIA	7	11.7
UGI BLEED	4	6.7
VOMITING	1	1.7

BREATHLESSNESS

FEVER

FEVER HEADACHE

FEVER,BODY STIFFNESS

FEVER,DYSURIA

FEVER,HIGH COLORED
URINE

INCREASED SALIVATION

INCREASED
SWEATING,SALIVATION

JAUNDICE

NO SYMPTOMS

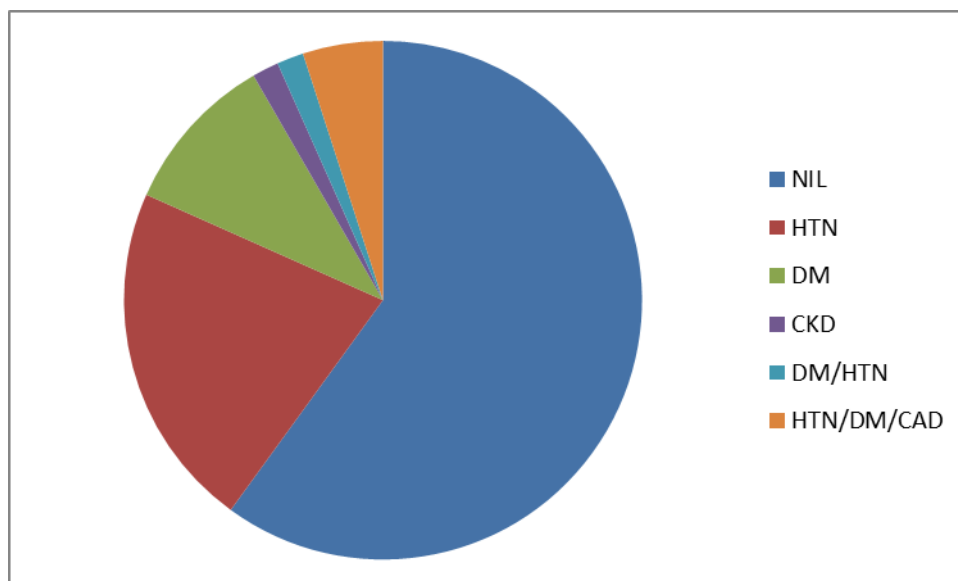
TIA

UGI BLEED

VOMITING

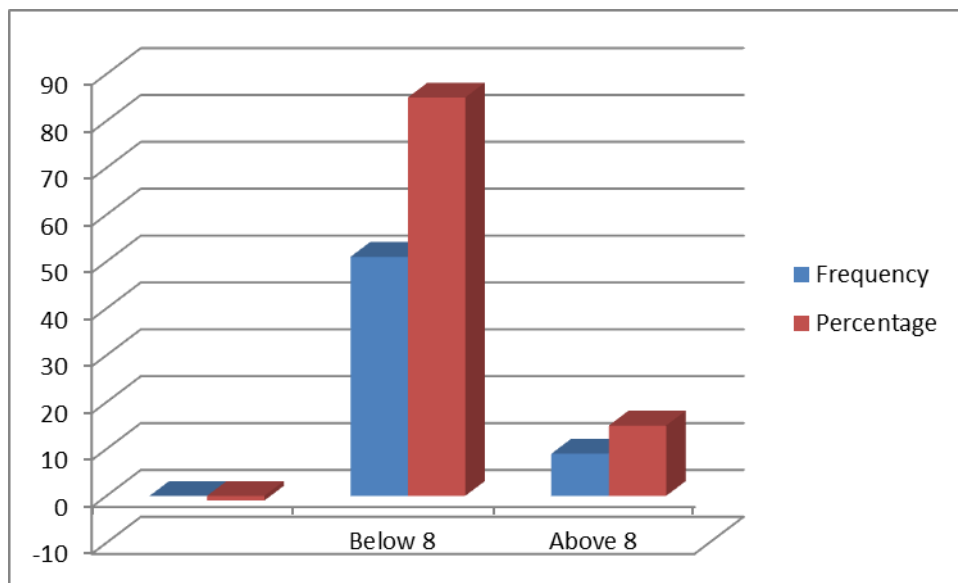
RISK FACTORS(1e)

Particulars	Frequency (n=60)	Percentage -100%
NIL	36	60
HTN	13	21.7
DM	6	10
CKD	1	1.7
DM/HTN	1	1.7
HTN/DM/CAD	3	5



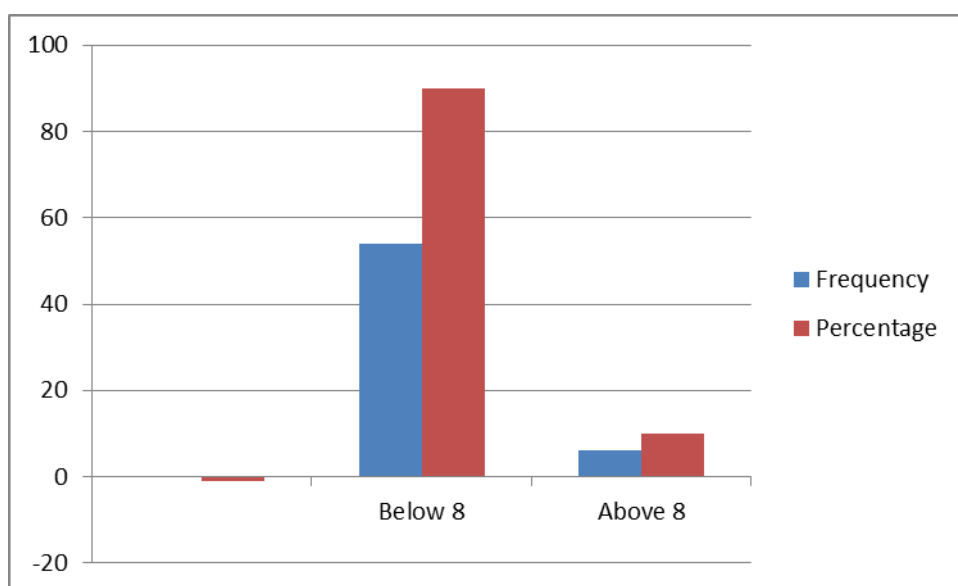
GCS AT ADMISSION(1f)

Particulars	Frequency (n=60)	Percentage -100%
Below 8	51	85
Above 8	9	15



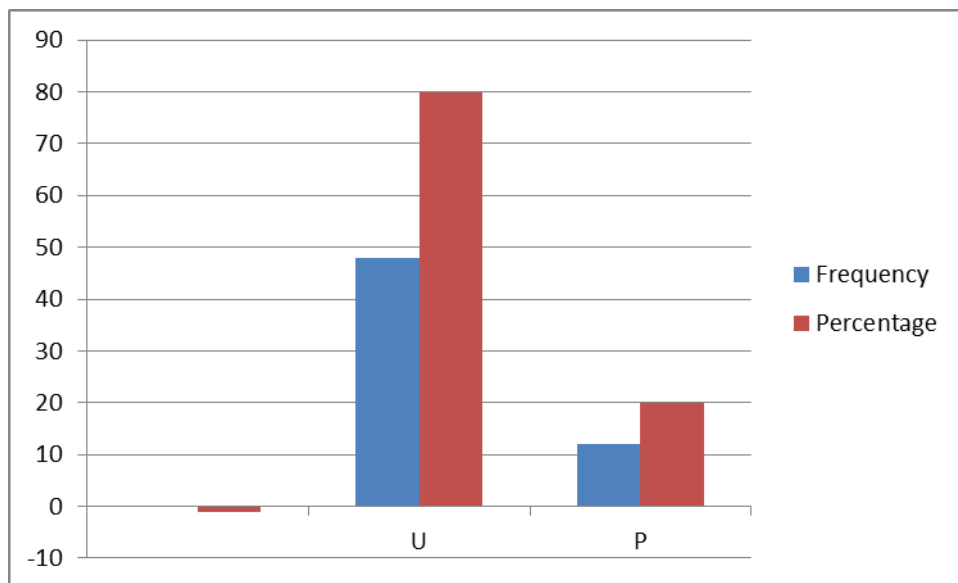
GCS AT 24 HOURS(1g)

Particulars	Frequency (n=60)	Percentage -100%
Below 8	54	90
Above 8	6	10



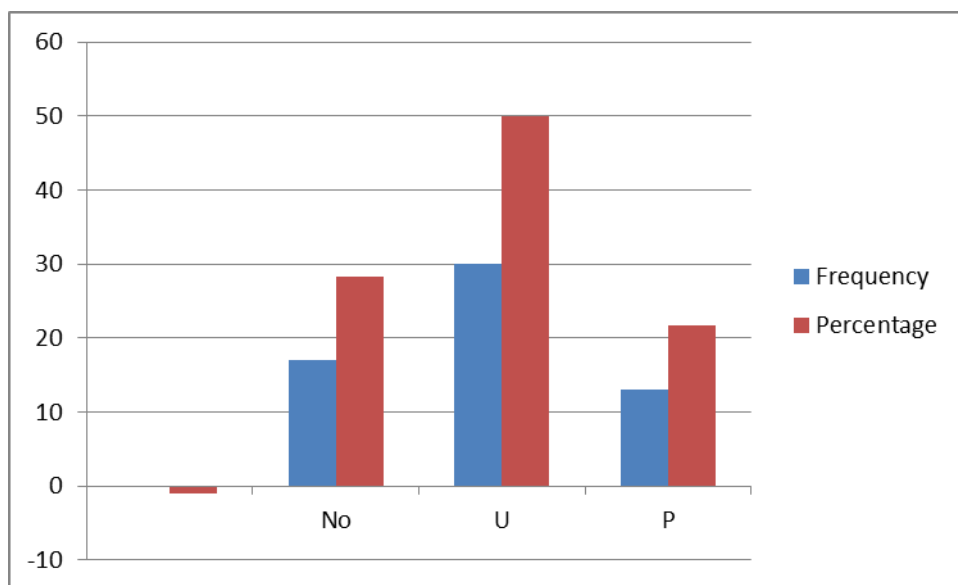
AVPU AT ADMISSION(1h)

Particulars	Frequency (n=60)	Percentage -100%
U	48	80
P	12	20



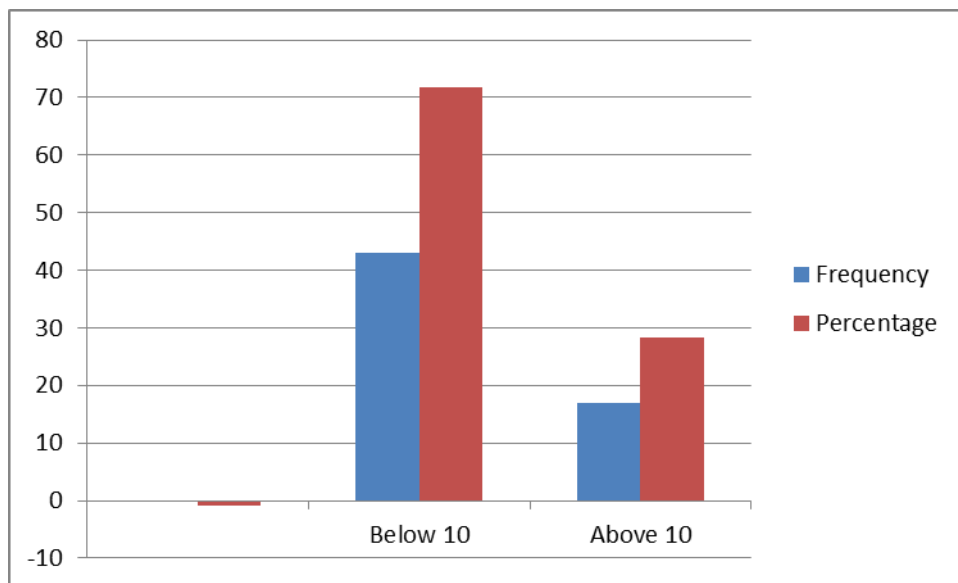
AVPU AT 24 HOURS(1i)

Particulars	Frequency (n=60)	Percentage -100%
No	17	28.3
U	30	50
P	13	21.7



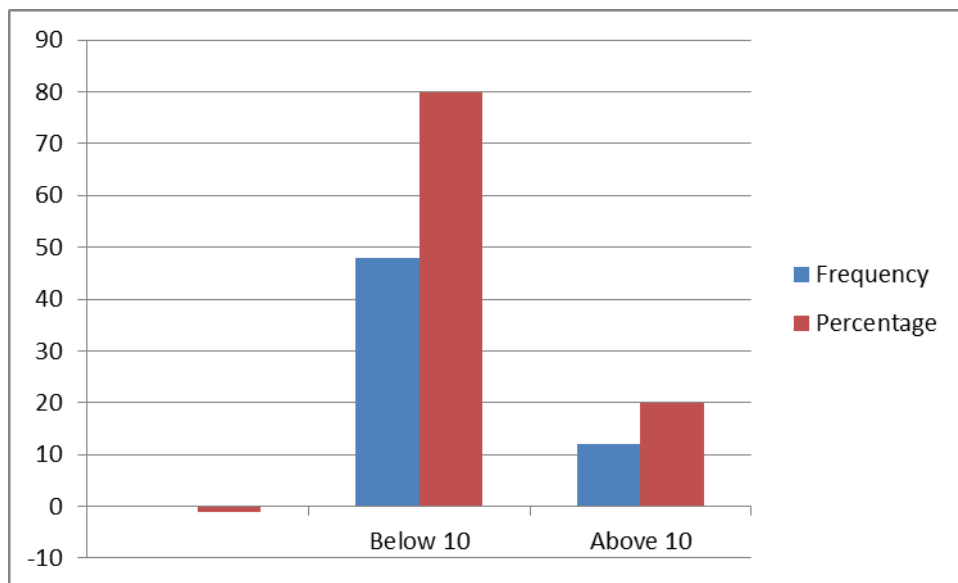
FSCS AT ADMISSION(1 j)

Particulars	Frequency (n=60)	Percentage -100%
Below 10	43	71.7
Above 10	17	28.3



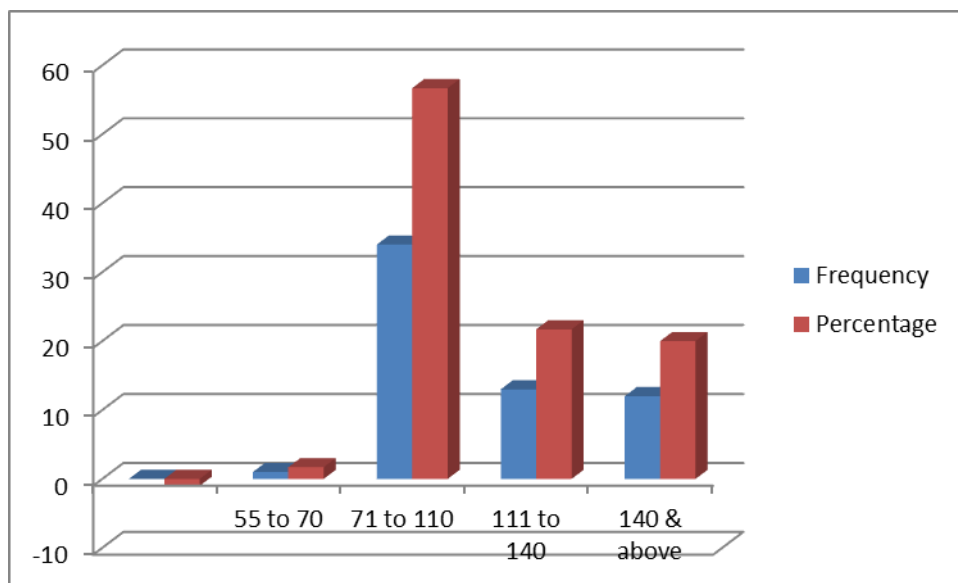
FSCS AT 24 HOURS(1 k)

Particulars	Frequency (n=60)	Percentage -100%
Below 10	48	80
Above 10	12	20



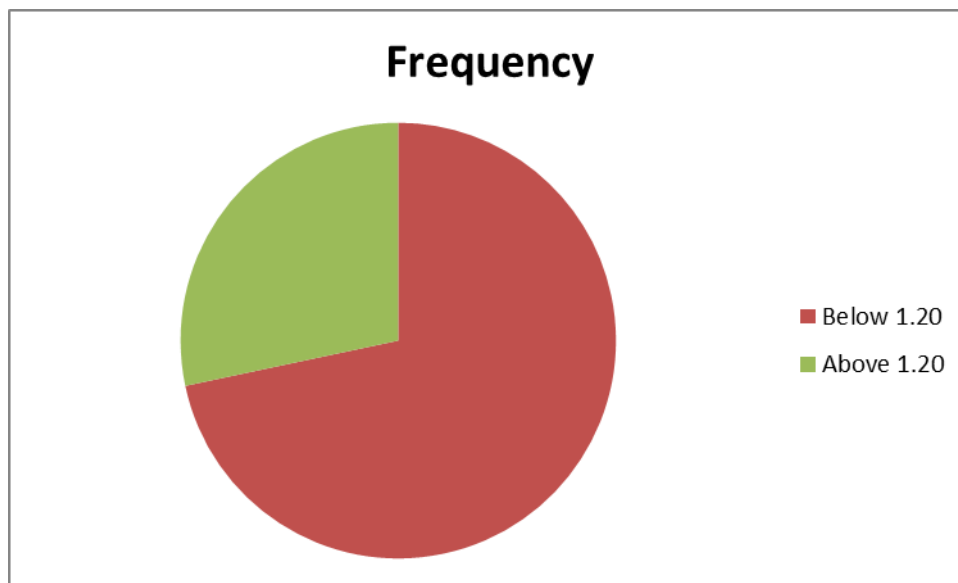
RBS(1 I)

Particulars	Frequency (n=60)	Percentage -100%
55 to 70	1	1.7
71 to 110	34	56.7
111 to 140	13	21.7
140 & above	12	20



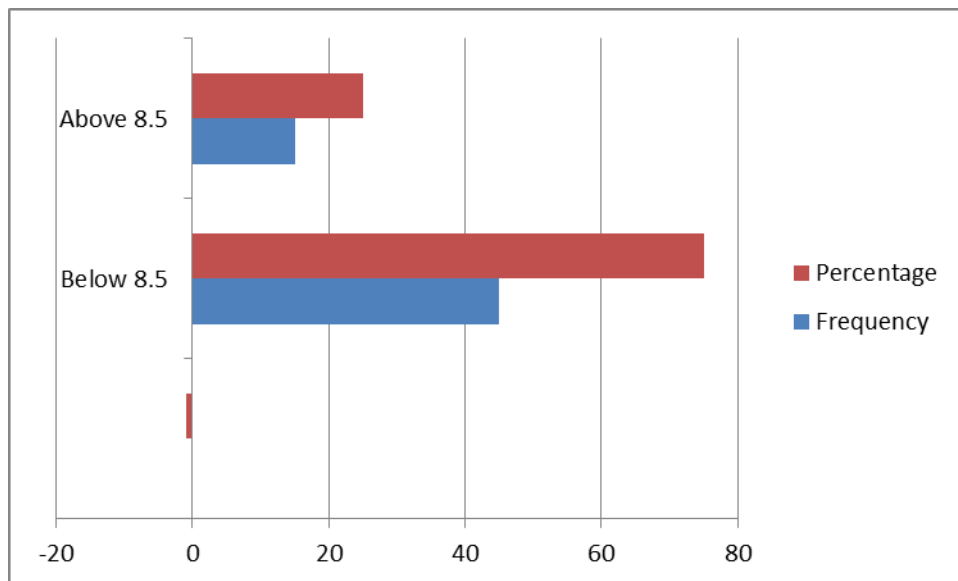
S.CREATITINE(1 m)

Particulars	Frequency (n=60)	Percentage -100%
Below 1.20	43	71.7
Above 1.20	17	28.3



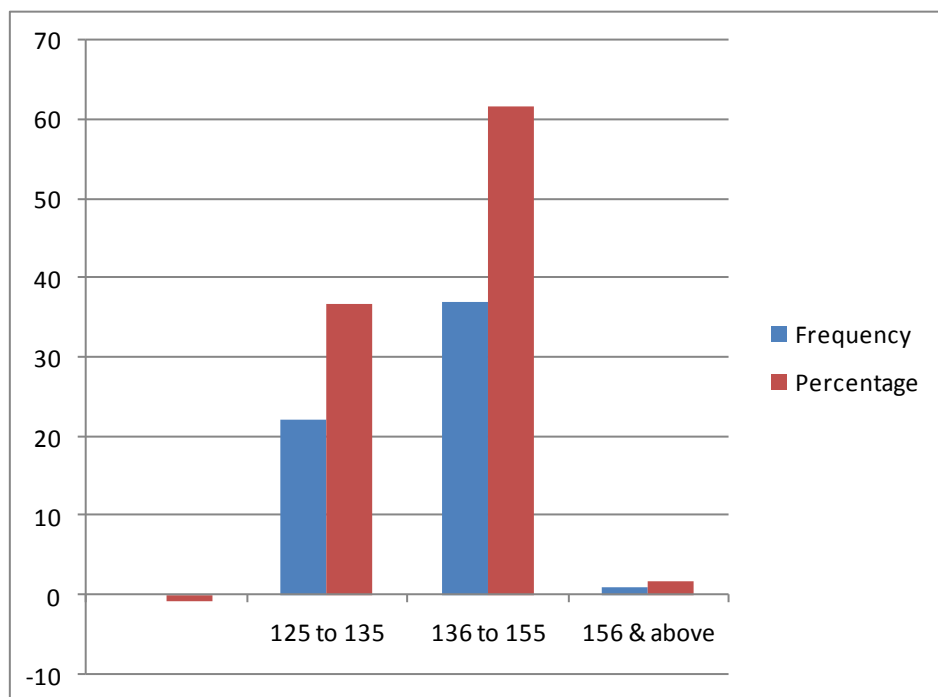
S CALCIUM(1n)

Particulars	Frequency (n=60)	Percentage (100%)
Below 8.5	45	75.0
Above 8.5	15	25.0



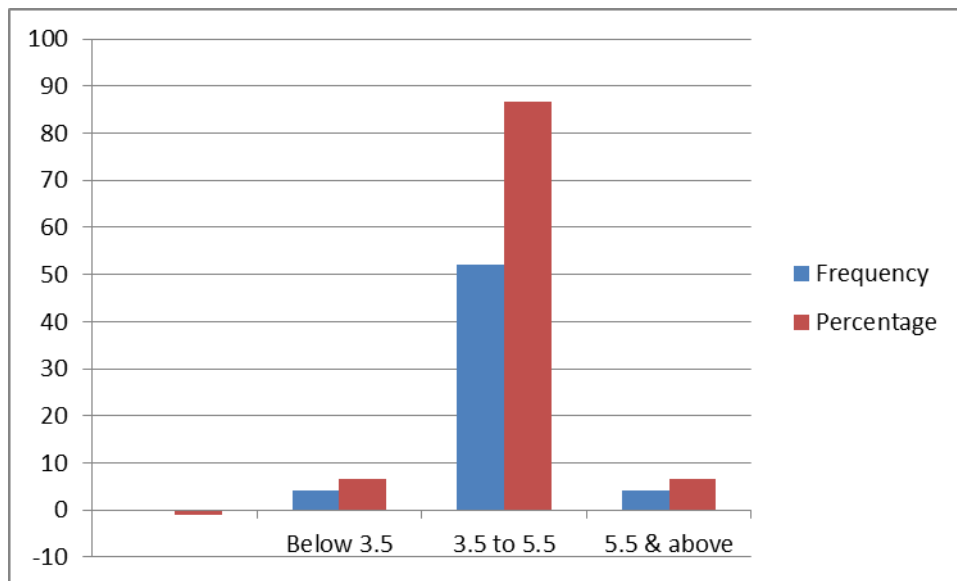
S.SODIUM(1o)

Particulars	Frequency (n=60)	Percentage -100%
125 to 135	22	36.7
136 to 155	37	61.7
156 & above	1	1.7



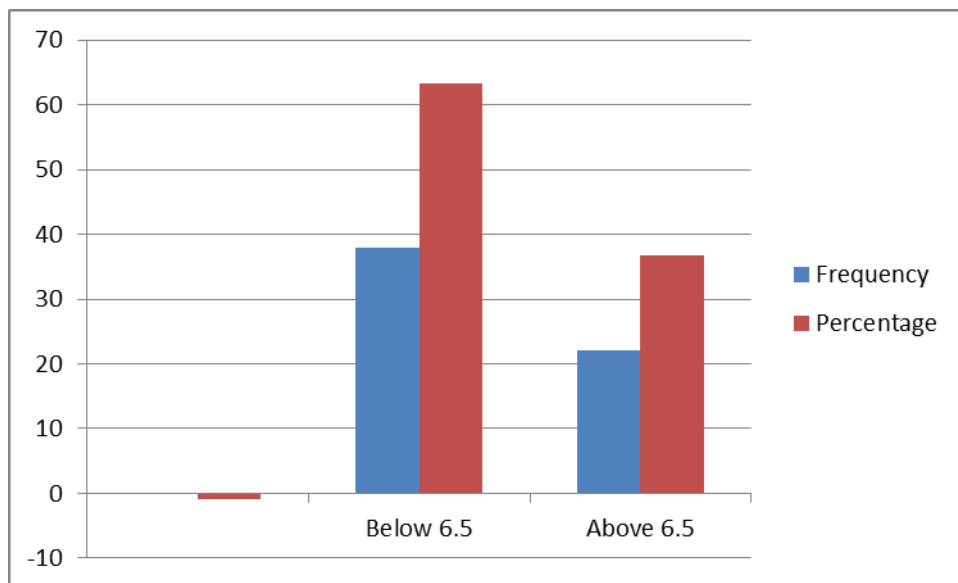
S.POTASSIUM(1 p)

Particulars	Frequency (n=60)	Percentage -100%
Below 3.5	4	6.7
3.5 to 5.5	52	86.7
5.5 & above	4	6.7



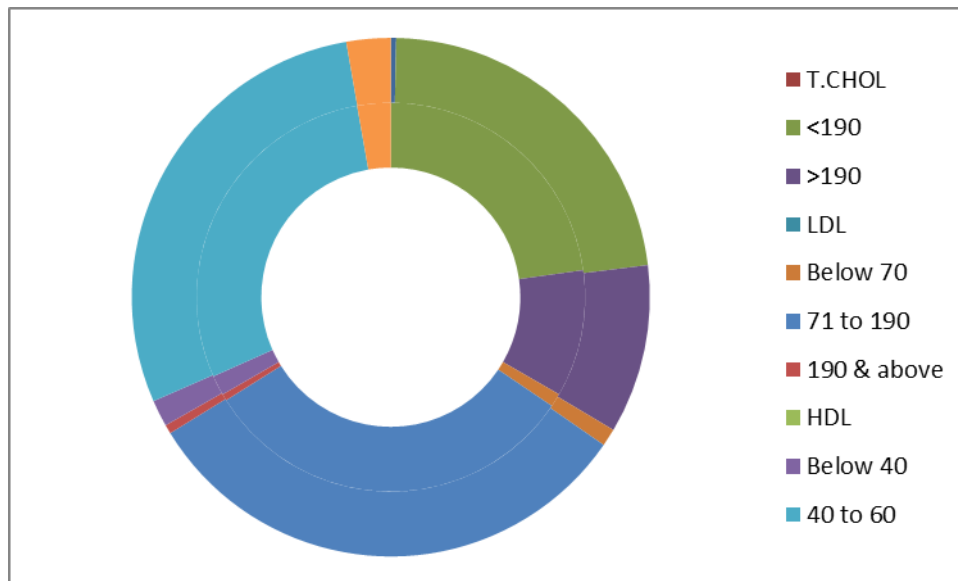
TOTAL PROTEIN(1 q)

Particulars	Frequency (n=60)	Percentage -100%
Below 6.5	38	63.3
Above 6.5	22	36.7



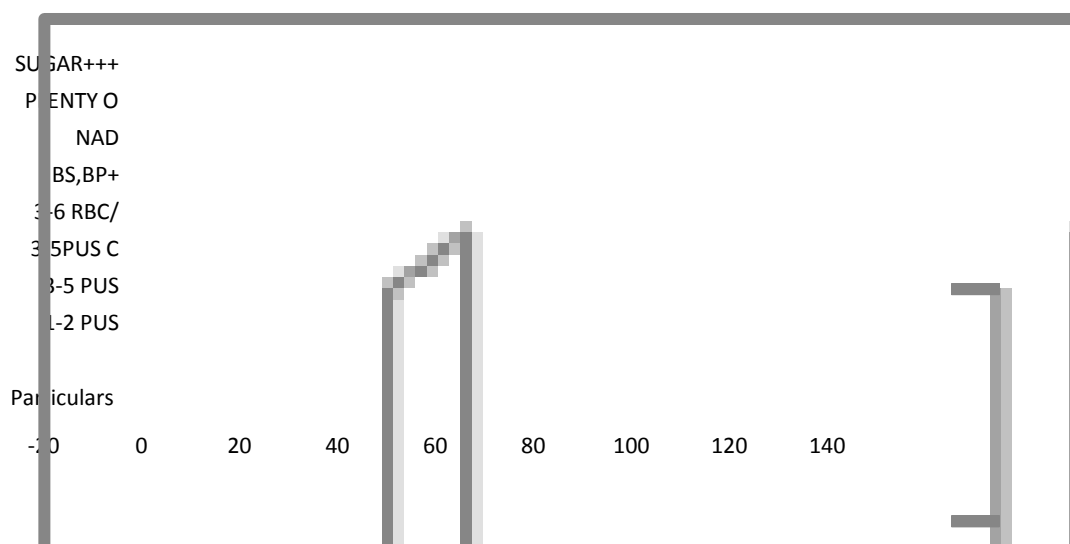
LIPID PROFILE(1 r)

Particulars	Frequency (n=60)	Percentage -100%
T.CHOL		
<190	41	68.3
>190	19	31.7
LDL		
Below 70	2	3.3
71 to 190	57	95
190 & above	1	1.7
HDL		
Below 40	3	5
40 to 60	52	86.7
60 & above	5	8.3



URINE ROUTINE(1s)

Particulars	Frequency (n=60)	Percentage (100%)
1-2 PUS	1	1.7
3-5 PUS	1	1.7
3-5PUS C	1	1.7
3-6 RBC/	1	1.7
BS,BP+	2	3.3
NAD	51	85.0
PLENTY O	2	3.3
SUGAR+++	1	1.7



ECG(1 t)

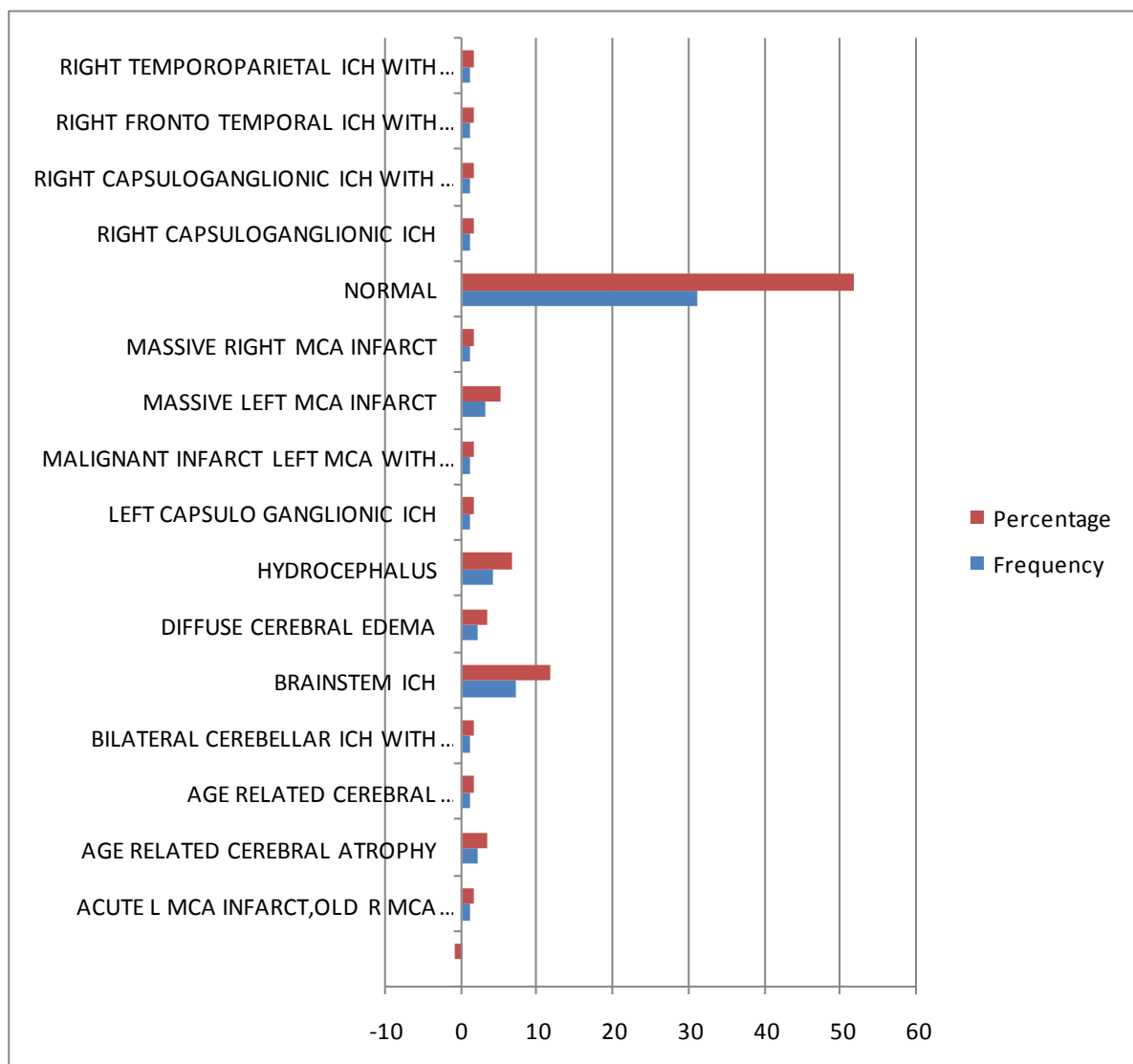
Particulars	Frequency (n=60)	Percentage (100%)
LVH	5	8.3
LVH WITH	1	1.7
NSR	47	78.3
S.BRADYC	2	3.4
S TACHYC	5	8.3

Frequency

LVH
LVH WITH STRAIN
NSR
S.BRADY
S TACHYC

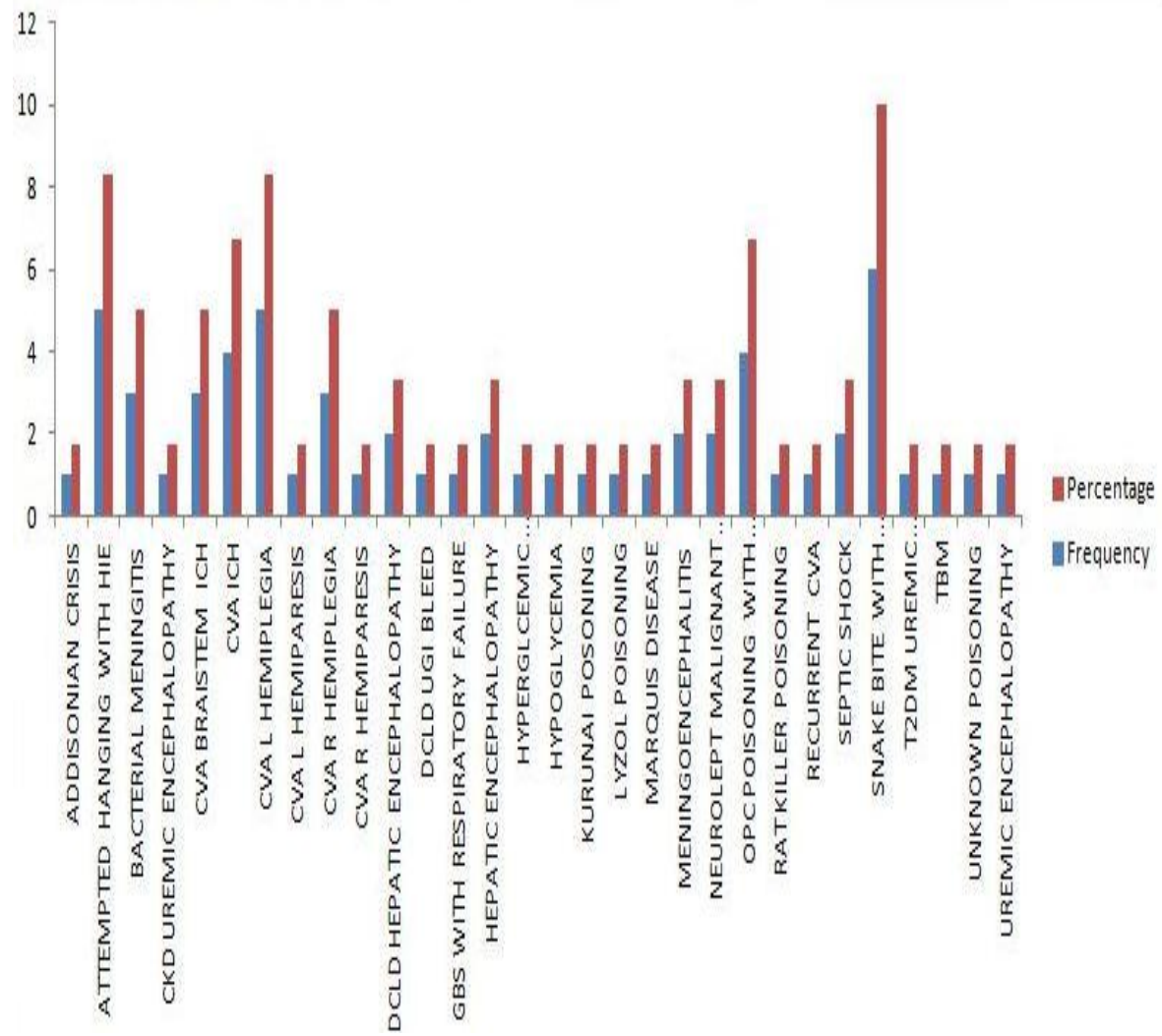
CT BRAIN (PLAIN) (1 u)

Particulars	Frequency (n=60)	Percentage -100%
ACUTE L MCA INFARCT,OLD R MCA INFARCT	1	1.7
AGE RELATED CEREBRAL ATROPHY	2	3.3
AGE RELATED CEREBRAL ATROPHY,HYDROCEPHALUS	1	1.7
BILATERAL CEREBELLAR ICH WITH BRAINSTEM ICH	1	1.7
BRAINSTEM ICH	7	11.7
DIFFUSE CEREBRAL EDEMA	2	3.3
HYDROCEPHALUS	4	6.7
LEFT CAPSULO GANGLIONIC ICH	1	1.7
MALIGNANT INFARCT LEFT MCA WITH MASS EFFECT	1	1.7
MASSIVE LEFT MCA INFARCT	3	5
MASSIVE RIGHT MCA INFARCT	1	1.7
NORMAL	31	51.7
RIGHT CAPSULOGANGLIONIC ICH	1	1.7
RIGHT CAPSULOGANGLIONIC ICH WITH IVE	1	1.7
RIGHT FRONTO TEMPORAL ICH WITH IVE;SAH	1	1.7
RIGHT TEMPOROPARIETAL ICH WITH IVE,HYDROCEPHALUS	1	1.7



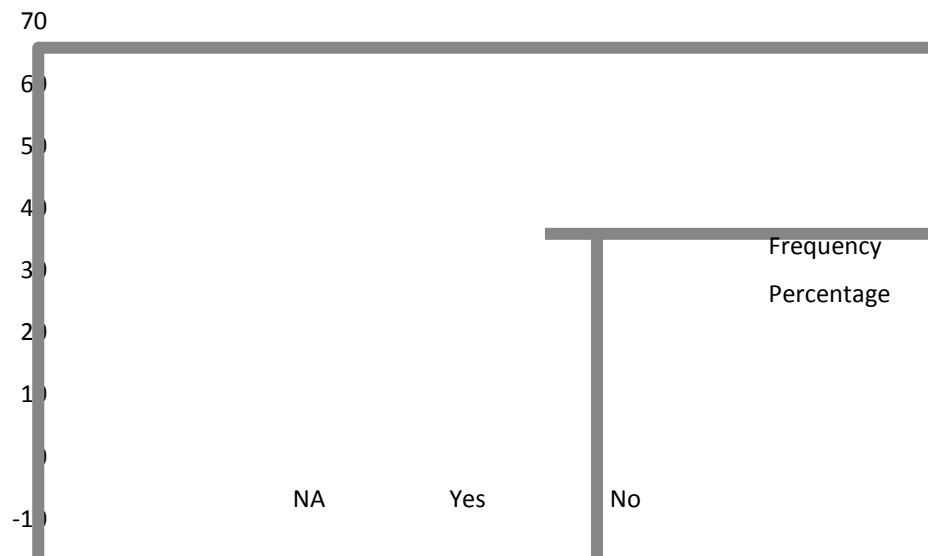
DIAGNOSIS(1 v)

Particulars	Frequency (n=60)	Percentage -100%
ADDISONIAN CRISIS	1	1.7
ATTEMPTED HANGING WITH HIE	5	8.3
BACTERIAL MENINGITIS	3	5
CKD UREMIC ENCEPHALOPATHY	1	1.7
CVA BRAISTEM ICH	3	5
CVA ICH	4	6.7
CVA L HEMIPLEGIA	5	8.3
CVA L HEMIPARESIS	1	1.7
CVA R HEMIPLEGIA	3	5
CVA R HEMIPARESIS	1	1.7
DCLD HEPATIC ENCEPHALOPATHY	2	3.3
DCLD UGI BLEED	1	1.7
GBS WITH RESPIRATORY FAILURE	1	1.7
HEPATIC ENCEPHALOPATHY	2	3.3
HYPERGLCEMIC HYPEROSMOTIC STATE	1	1.7
HYPOGLYCEMIA	1	1.7
KURUNAI POSONING	1	1.7
LYZOL POISONING	1	1.7
MARQUIS DISEASE	1	1.7
MENINGOENCEPHALITIS	2	3.3
NEUROLEPT MALIGNANT SYNDROME	2	3.3
OPC POISONING WITH RESP.FAILURE	4	6.7
RAT KILLER POISONING	1	1.7
RECURRENT CVA	1	1.7
SEPTIC SHOCK	2	3.3
SNAKE BITE WITH NEUROPARALYSIS	6	10
T2DM UREMIC ENCEPHALOPATHY	1	1.7
TBM	1	1.7
UNKNOWN POISONING	1	1.7
UREMIC ENCEPHALOPATHY	1	1.7



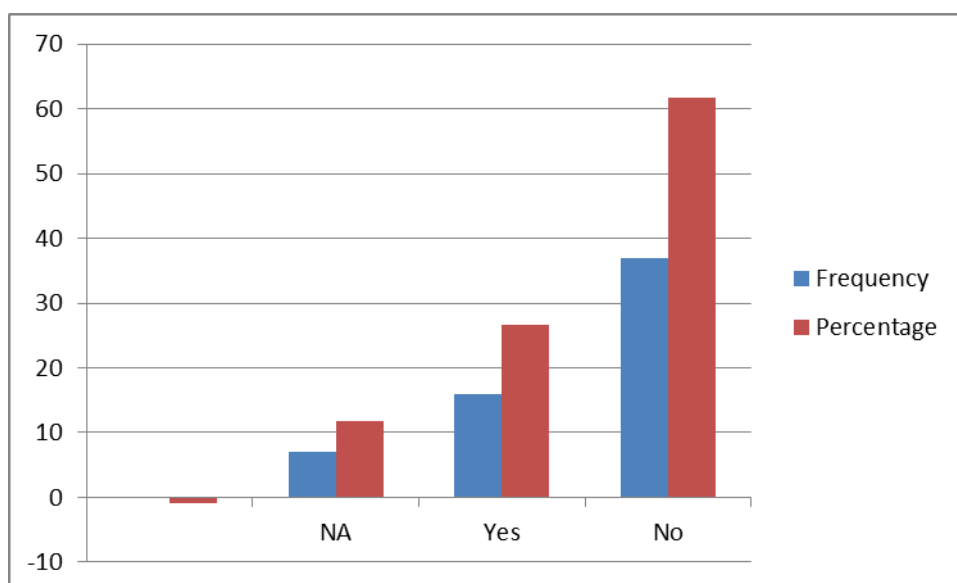
MORTALITY WITHIN 24 HOURS(1 w)

Particulars	Frequency (n=60)	Percentage -100%
NA	7	11.7
Yes	16	26.7
No	37	61.7



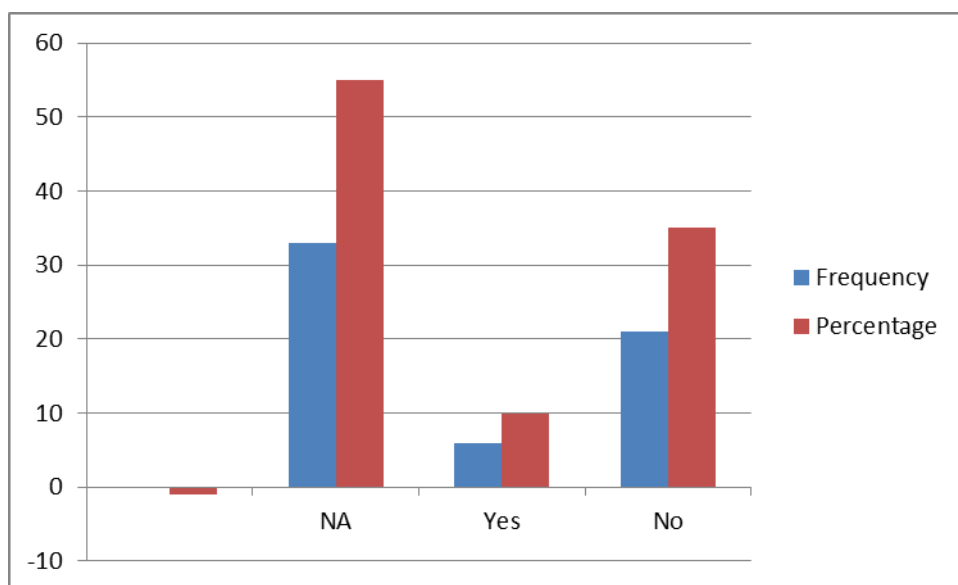
MORTALITY 24-48 HOURS(1 x)

Particulars	Frequency (n=60)	Percentage -100%
NA	7	11.7
Yes	16	26.7
No	37	61.7



MORTALITY 48-72 HOURS(1 y)

Particulars	Frequency (n=60)	Percentage -100%
NA	33	55
Yes	6	10
No	21	35



Descriptive Statistics

Item	Min.	Max.	Mean	S.D
q1.AGE	19	80	47.95	16.857
q11.GCS SCORE (AD)	3	11	5.48	2.411
q11.GCS SCORE (AT 24 HRS)	0	14	4.15	3.621
q13.FSCS SCORE(AD)	1	15	7.90	3.917
q13.FSCS SCORE(AT 24 HRS)	0	15	5.53	5.027
q14.RBS	55	632	122.88	80.086
q15.UREA	16	249	52.18	38.265
q16.CREATININE	.60	14.80	1.7483	2.51770
q17.SODIUM	126	156	137.78	6.982
q18.POTASSIUM	3.10	7.60	4.2017	.76057
q19.CALCIUM	5.00	10.10	7.6267	1.17587
q21.LFT (SGOT)	33	676	85.02	126.847
q22.LFT(SGPT)	21	1787	91.27	239.135
q23.LFT (T.PR)	5.00	9.00	6.3950	.71365

LIPID PROFILE-T.CHOL	165	398	194.32	31.783
LIPID PROFILE- LDL	67	194	103.67	27.522
LIPID PROFILE- HDL	35	67	50.33	7.271

Chi-square test (3a)

	Age										Statistical inference
	13 to 33		34 to 54		55 to 75		76 & above		Total		
	(n=1)	(100%)	(n=24)	(100%)	(n=20)	(100%)	(n=3)	(100%)	(n=60)	(100%)	
q27.DIAGNOSIS											
ADDISONI	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	X ² =80.974 Df=87 .662>0.05 Not Significant
ATTEMPTED HANGING	3	23.1%	0	.0%	2	10.0%	0	.0%	5	8.3%	
BACTERIAL MENINGITIS	1	7.7%	0	.0%	2	10.0%	0	.0%	3	5.0%	
CKD UREMIC ENCEPHALOPATHY	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	
CVA-BRAINSTEM ICH	0	.0%	1	4.2%	2	10.0%	0	.0%	3	5.0%	
CVA ICH	0	.0%	2	8.3%	1	5.0%	1	33.3%	4	6.7%	
CVA L HEMIPLEGIA	1	7.7%	2	8.3%	1	5.0%	1	33.3%	5	8.3%	
CVA L HEMIPARESIS	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	
CVA R HEMIPLEGIA	0	.0%	2	8.3%	1	5.0%	0	.0%	3	5.0%	
CVA R HEMIPARESIS	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	
DCLD HEPATIC ENCEPHALOPATHY	0	.0%	1	4.2%	1	5.0%	0	.0%	2	3.3%	
DCLD/UGI BLEED	0	.0%	1	4.2%	0	.0%	0	.0%	1	1.7%	
GBS WITH RESPIRATORY FAILURE	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	
HEPATIC ENCEPHALOPATHY	1	7.7%	0	.0%	1	5.0%	0	.0%	2	3.3%	
HYPERGLYCEMIC HYPEROSMOLAR STATE	0	.0%	1	4.2%	0	.0%	0	.0%	1	1.7%	
HYPOGLYCEMIA	0	.0%	0	.0%	1	5.0%	0	.0%	1	1.7%	

[illegible]

NA	2	15.4%	2	8.3%	3	15.0%	0	.0%	7	11.7%	X ² =3.990 Df=6 .678>0.05 Not Significant
Yes	5	38.5%	4	16.7%	6	30.0%	1	33.3%	16	26.7%	
No	6	46.2%	18	75.0%	11	55.0%	2	66.7%	37	61.7%	
Mortality 24 to 48hrs											
NA	7	53.8%	7	29.2%	9	45.0%	1	33.3%	24	40.0%	X ² =13.533 Df=6 .035<0.05 Significant
Yes	5	38.5%	3	12.5%	1	5.0%	0	.0%	9	15.0%	
No	1	7.7%	14	58.3%	10	50.0%	2	66.7%	27	45.0%	
Mortality 48 to 72hrs											
NA	12	92.3%	10	41.7%	10	50.0%	1	33.3%	33	55.0%	X ² =12.694 Df=6 .048<0.05 Significant
Yes	1	7.7%	3	12.5%	1	5.0%	1	33.3%	6	10.0%	
No	0	.0%	11	45.8%	9	45.0%	1	33.3%	21	35.0%	
q11.GCS SCORE (AD)											
Below 8	10	76.9%	21	87.5%	17	85.0%	3	100.0%	51	85.0%	X ² =1.312 Df=3 .04726<0.05 Significant
Above 8	3	23.1%	3	12.5%	3	15.0%	0	.0%	9	15.0%	
q11.GCS SCORE; AT 24HRS											
Below 8	12	92.3%	22	91.7%	17	85.0%	3	100.0%	54	90.0%	X ² =1.040 Df=3 .792>0.05 Not Significant
Above 8	1	7.7%	2	8.3%	3	15.0%	0	.0%	6	10.0%	

q12. AVPU SCORE AD											
U	11	84.6%	19	79.2%	15	75.0%	3	100.0%	48	80.0%	$X^2=1.246$ Df=3 .042<0.05 Significant
P	2	15.4%	5	20.8%	5	25.0%	0	.0%	12	20.0%	
q12. AVPU SCORE AT 24HRS											
No	4	30.8%	6	25.0%	6	30.0%	1	33.3%	17	28.3%	$X^2=3.411$ Df=6 .756>0.05 Not Significant
U	8	61.5%	11	45.8%	9	45.0%	2	66.7%	30	50.0%	
P	1	7.7%	7	29.2%	5	25.0%	0	.0%	13	21.7%	
q13.FSCS SCORE AD											
Below 10	10	76.9%	17	70.8%	14	70.0%	2	66.7%	43	71.7%	$X^2=.249$ Df=3 .969>0.05 Not Significant
Above 10	3	23.1%	7	29.2%	6	30.0%	1	33.3%	17	28.3%	
q13.FSCS SCORE AT 24HRS											
Below 10	12	92.3%	20	83.3%	14	70.0%	2	66.7%	48	80.0%	$X^2=2.981$ Df=3 .0395<0.05 Significant
Above 10	1	7.7%	4	16.7%	6	30.0%	1	33.3%	12	20.0%	

Chi-square test (3b)

Risk factors	T.CHOL						Statistica l inference
	<190		>190		Total		
	(n=41)	(100%)	(n=19)	(100%)	(n=60)	(100%)	
Nil	34	82.9%	2	10.5%	36	60.0%	X ² =37.05 1 Df=5 .000<0.05 Significa nt
HTN	5	12.2%	8	42.1%	13	21.7%	
DM	0	.0%	6	31.6%	6	10.0%	
CKD	1	2.4%	0	.0%	1	1.7%	
HTN,CKD	1	2.4%	0	.0%	1	1.7%	
HTN,DM,CA D	0	.0%	3	15.8%	3	5.0%	

Chi-square test(3c)

Risk factors	LDL								Statistical inference
	Below 70		71 to 190		190 & above		Total		
	(n=2)	(100%)	(n=57)	(100%)	(n=1)	(100%)	(n=60)	(100%)	
Nil	2	100.0%	34	59.6%	0	.0%	36	60.0%	X ² =20.643 Df=10 .024<0.05 Significant
HTN	0	.0%	13	22.8%	0	.0%	13	21.7%	
DM	0	.0%	6	10.5%	0	.0%	6	10.0%	
CKD	0	.0%	1	1.8%	0	.0%	1	1.7%	
HTN,CKD	0	.0%	1	1.8%	0	.0%	1	1.7%	
HTN,DM,CAD	0	.0%	2	3.5%	1	100.0%	3	5.0%	

Chi-square test(3d)

Risk factors	HDL								Statistical inference
	Below 40		40 to 60		60 & above		Total		
	(n=3)	(100%)	(n=52)	(100%)	(n=5)	(100%)	(n=60)	(100%)	
Nil	3	100.0%	29	55.8%	4	80.0%	36	60.0%	X ² =3.685 Df=10 .960>0.05 Not Significant
HTN	0	.0%	12	23.1%	1	20.0%	13	21.7%	
DM	0	.0%	6	11.5%	0	.0%	6	10.0%	
CKD	0	.0%	1	1.9%	0	.0%	1	1.7%	
HTN,CKD	0	.0%	1	1.9%	0	.0%	1	1.7%	
HTN,DM,CAD	0	.0%	3	5.8%	0	.0%	3	5.0%	

Chi square test(3e)

	DURATION OF ILLNESS										Statistical inference
	Below 6hrs		6 to 24hrs		24 to 48hrs		48hrs & above		Total		
	(n=27)	(100%)	(n=11)	(100%)	(n=11)	(100%)	(n=11)	(100%)	(n=60)	(100%)	
Mortality with in 24hrs											
NA	1	3.7%	2	18.2%	2	18.2%	2	18.2%	7	11.7%	X ² =4.331 Df=6 .032<0.05 Significant
Yes	8	29.6%	4	36.4%	2	18.2%	2	18.2%	16	26.7%	
No	18	66.7%	5	45.5%	7	63.6%	7	63.6%	37	61.7%	
Mortality 24 to 48hrs											
NA	10	37.0%	6	54.5%	4	36.4%	4	36.4%	24	40.0%	X ² =8.724 Df=6 .019<0.05 Significant
Yes	3	11.1%	2	18.2%	0	.0%	4	36.4%	9	15.0%	
No	14	51.9%	3	27.3%	7	63.6%	3	27.3%	27	45.0%	
Mortality 48 to 72hrs											
NA	13	48.1%	8	72.7%	4	36.4%	8	72.7%	33	55.0%	X ² =10.651 Df=6 .100>0.05 Not Significant
Yes	4	14.8%	2	18.2%	0	.0%	0	.0%	6	10.0%	
No	10	37.0%	1	9.1%	7	63.6%	3	27.3%	21	35.0%	
q27.DIAGNOSIS											
ADDISONI	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%	X ² =115.421Df=87 .022<0.05 Significant
ATTEMPTTE	4	14.8%	1	9.1%	0	.0%	0	.0%	5	8.3%	
BACTERIA	1	3.7%	0	.0%	0	.0%	2	18.2%	3	5.0%	
CKD UREM	0	.0%	1	9.1%	0	.0%	0	.0%	1	1.7%	
CVA-BRAINSTEM ICH	2	7.4%	1	9.1%	0	.0%	0	.0%	3	5.0%	
CVA ICH	2	7.4%	1	9.1%	1	9.1%	0	.0%	4	6.7%	

CVA L HEMIPLEGIA	3	11.1%	0	.0%	2	18.2%	0	.0%	5	8.3%
CVA L HEMIPARESIS	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%
CVA R HEMIPLEGIA	1	3.7%	1	9.1%	1	9.1%	0	.0%	3	5.0%
CVA R HEMIPARESIS	0	.0%	0	.0%	1	9.1%	0	.0%	1	1.7%
DCLD HEP ENCEPH	0	.0%	0	.0%	0	.0%	2	18.2%	2	3.3%
DCLD/UGI BLEED	0	.0%	0	.0%	1	9.1%	0	.0%	1	1.7%
GBS WITH RESP FAILURE	0	.0%	0	.0%	0	.0%	1	9.1%	1	1.7%
HEPATIC ENCEPHALOPATHY	0	.0%	0	.0%	0	.0%	2	18.2%	2	3.3%
HYPERGLYCEMIC HYPEROSMOLAR STATE	0	.0%	1	9.1%	0	.0%	0	.0%	1	1.7%
HYPOGLYCEMIA	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%
KURUNAI POISONING	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%
LYZOL POISONING	0	.0%	1	9.1%	0	.0%	0	.0%	1	1.7%
MARQUIS DISEASE	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%
MENINGOENCEPHALITIS	0	.0%	0	.0%	0	.0%	2	18.2%	2	3.3%
NEUROLEPT MALIGNANT SYNDROME	0	.0%	0	.0%	2	18.2%	0	.0%	2	3.3%
OPC POISONING WITH RESP FAILURE	3	11.1%	1	9.1%	0	.0%	0	.0%	4	6.7%
RAT KILLER PASTE POISONING	1	3.7%	0	.0%	0	.0%	0	.0%	1	1.7%
RECURRENT CVA	0	.0%	0	.0%	1	9.1%	0	.0%	1	1.7%
SEPTIC SHOCK	1	3.7%	0	.0%	1	9.1%	0	.0%	2	3.3%
SNAKE BITE	4	14.8%	2	18.2%	0	.0%	0	.0%	6	10.0%

T2DM/UROSEPSIS	0	.0%	0	.0%	0	.0%	1	9.1%	1	1.7%	
TBM	0	.0%	0	.0%	0	.0%	1	9.1%	1	1.7%	
UNKNOWN POISONING RESP.FAILURE	0	.0%	1	9.1%	0	.0%	0	.0%	1	1.7%	
UREMIC ENCEPHALOPATHY	0	.0%	0	.0%	1	9.1%	0	.0%	1	1.7%	

DISCUSSION

DISCUSSION

This study was conducted to find out the causes of coma in non head injury cases in our part of the country, correlation with duration of illness and mortality; correlation with various scores and mortality, CT brain findings, diagnosis and mortality and usefulness of various scores in assessing the short term prognosis. Since there are not much studies about adult non traumatic coma, this study will be useful in finding out the common causes of medical coma in this part of our country.

60 adults presented to MGM GH attached to KAPV Govt Medical College in an unconscious state was the study population. Biochemical, Hematopathological and imaging studies were done for all cases to find out the cause and scores were given at admission and 24, 48, 72 hours to assess prognosis

AGE

Middle and old age patients were more affected than young adults³¹ (78.3% against 21.7%). Old age patients with coma had CVA as common cause while young adults presented with poisoning or attempted hanging. Infections were common for both groups and bacterial meningitis and tuberculous meningitis^{35 36 31}

SEX

Male preponderance was there (61.7%) similar to other studies^{31 32}

DURATION OF ILLNESS

In 45% of cases short duration of illness was noted(< 6 hrs),the rest all-namely 6-24hrs,24-48 hrs,>48 hrs showed equal percentage³²

SYMPTOMS

40% were not having any symptoms preceeding or associated with the illness. 13% had breathlessness and 11.7 % had TIA

RISK FACTORS

1)LIPID PROFILE

- 54 % was found to be dyslipidemic during the study.
- Total cholesterol was in high normal range in 13 patients(21.6%)
- 11 had high normal T.Cholesterol(18.3%)
- T.cholesterol was found to be stastically significant (p value-.000)
- 22 out of 60 had elevated LDL cholesterol(36.6%)
- LDL was statistically significant(p value-.024)
- HDL cholesterol was found to be in normal range
- HDL was not statistically significant(p value-.960)

2)HYPERTENSION/DIABETES/CAD/CKD

- 13 out of 60 were known hypertensives(21.7%)
- All hypertensives were having impaired lipid profile in the form of high normal T.Cholesterol or high LDL
- 6 had diabetes(10%)
- All diabetic patients were invariably dyslipidemics;had elevated T.Cholesterol

- 1 was CKD patient. 1 had Hypertension and CKD and 3 were having Hypertension, CKD and CAD.
- CAD patients also had high T. Cholesterol and HDL
- Those with hypertension as risk factor had predilection for stroke; more for ICH as seen in other studies³¹

GCS Score:

85% were having scores < 8 at admission and 90% were having < 8 score after 24 hours. Patients with low score had poor prognosis as compared to other studies^{31 32 33}.

18 patients with GCS less than or equal to 8 expired within 24 hours of admission. GCS at admission less than 8 was statistically significant³⁴ (p-value: 0.047)

AVPU score

80 % were unconscious at admission and out of that, 28 % expired at 24 hours. 50 % of the remaining were unconscious at 24 hrs. 17 patients who were unconscious at admission expired within 24 hours. AVPU score at admission was statistically significant (p value: 0.042)

FSCS Score

72% had a total score below 10 at admission and 80% had score below 10 at 24 hours. FSCS at admission was not statistically significant but score below 10 at 24 hours was significant (p value: 0.0395)

RBS

Only 1 person had Hypoglycemia and 12 patients had RBS more than 140. All the rest were in normal range. Hypoglycemic patient had an RBS of 55 g, and she regains consciousness after 25% dextrose infusion. One patient had Hyperglycemic Hyperosmolar state with an RBS of 632 g.

S.CREATININE

17 out of 60 participants had values more than 1.2 mg. Two patients were known CKD with uremic encephalopathy (S.Creatinine 14.8 and 10.1). Three patients following snake bite also had high creatinine values.

S.SODIUM

37 patients out of 60 had sodium in normal range. One had a value of 156 (CKD patient) and 22 had values less than 135 but none had values below 125. Hence hypernatremia and hyponatremia as a cause of metabolic coma was ruled out.

S.POTASSIUM

52 had normal potassium values, 4 had hyperkalemia and 4 had hypokalemia. None of the patients were symptomatic for hyper or hypokalemia.

S.CALCIUM

Even though calcium levels in the study population were 7.6, there was no clinical evidence of either hypocalcemia or hypercalcemia.

DIAGNOSIS

- Most common etiologic diagnosis was CVA-18 cases(30%) followed by poisoning-7 cases(11%) and snake bite -6 cases(10%)and attempted hanging 5 cases(8%)
- Of the CVA cases,haemorrhage was the leading cause-13 out of 18 were ICH(72%). In haemorrhage,there was 7 brainstem ICH,(53%) 3 capsuloganglionic ICH with Intra ventricular extension(IVE) 23%) 1 fronto temporal ICH with IVE and SAH;(7%) 1 bilateral cerebellar ICH with brainstem ICH (7%)1 temporoparietal ICH with IVE (7%)
- Remaining 5 CVA cases were Infarct;4 being massive MCA infarct and 1 being malignant infarct with mass effect OPC poisoning topped the list of poisoning, with 4 out of 7 were OPC poisoning with respiratory failure 1 case of Kurunai poisoning with respiratory failure,1 case of Lyzol poisoning with respiratory failure,1 Unknown tablet poisoning with respiratory failure and 1 Rat killer paste poisoning with fulminant hepatic failure there.
- 6 out of 60 cases were snake bite(10%) Three were with neuromuscular paralysis, two developed uremic encephalopathy following AKI;one had coagulopathy, manifesting as Brainstem ICH
- There were 5 cases of attempted hanging with Hypoxic ischemic encephalopathy

- Of the remaining coma cases,
 - Bacterial meningitis-4 cases
 - Hepatic encephalopathy stage 4-3 cases TBM-2
 - Urosepsis-2
 - Neurolept Malignant Syndrome-2
 - Hypoglycemia-1
 - Hyperglycemic Hyperosmolar state-1
 - MODS-1
 - Marquis disease-1
 - Addisonian crisis-1
 - GBS with respiratory failure-1

Diagnosis related to age was not statistically significant(p value-0.662)

Diagnosis related to duration of illness was statistically significant(p value-0.022)

MORTALITY

- 18 expired within 24 hours of admission(30%)
- 21 were alive after 72 hours and the rest expired within 24-72 hrs
- Out of all the immediate deaths,CVA with hemorrhagic stroke was most common cause in old age(3 out of 16 deaths) similar to other studies³¹
- Poisoning with respiratory failure(4 cases) and Snake bite with neuromuscular paralysis(2 cases) were the leading causes in young adults

- 2 cases of DCLD, 2 bacterial meningitis, one each of Urosepsis, Attempted hanging with HIE, GBS with Resp. Failure, Marquis disease and Addisonian Crisis succumbed to death within 24 hours of admission
- Of the ischemic causes of CVA, -4 out of 5 cases were alive after 72 hours of admission
- Only one case of Massive infarct expired within 48 hours
- Patients who were alive after 72 hours of admission were:
 - 1) CVA- 4 ischemic stroke, 4 hemorrhagic stroke
 - 2) Attempted hanging-3
 - 3) Neurolept Malignant Syndrome-2
 - 4) Hepatic encephalopathy-2
 - 5) Hypoglycemia-1
 - 6) OPC poisoning with Resp. failure-1
 - 7) Urosepsis-1
 - 8) MODS-1
 - 9) Uremic encephalopathy-1
 - 10) Snake bite with neuroparalysis-1

This shows that those who were alive after 72 hours of admission were mostly having non structural causes for coma.

Mortality related to age was not statistically significant for duration less than 24 hours (p value-0.678).

It was significant for duration 24-48 hours and 48-72 hours(p values:0.035 and 0.048 respectively).

Mortality within 24 hours related to duration of illness was statistically significant (p value 0.032).

Mortality between 24-48 hours in relation to duration of illness was significant(p value 0.019).

Mortality between 48-72 hours was not statistically significant(p value 0.100).

PROGNOSIS

Structural causes of coma had worst prognosis-CVA with ICH ; and the same is with non structural causes with severe CNS depression eg:OPC poisoning with respiratory failure.

Any disease causing global cerebral hypoxia and hypoperfusion even without a demonstrable structural lesion will cause coma and death within 24 hours³¹

CT BRAIN(PLAIN)

The metabolic, toxic and non structural causes for coma had normal imaging findings and other structural causes were identified from CT brain itself.

LIMITATIONS OF THE STUDY

- 1 Correlation with EEG is lacking
- 2 Duration of study is short
- 3 Sample size is small
- 4 Inability to correlate with higher imaging modality like MRI

SUMMARY

SUMMARY

- 1) The most common cause of non traumatic Coma in adults in our study is non structural brain disease.
- 2) The predominant cause of coma in
 - a) Young age-toxic and metabolic causes
 - b) Old age-cerebro vascular accident
 - c) Infections were noted in similar proportion in both age groups
- 3) CT brain showed abnormal finding in concordance with cause of coma in structural brain disease
- 4) CT brain did not show any demonstrable abnormality in most of the non structural causes of coma.
- 5) Hemorrhage is the most common finding in CT brain followed by Infarct. Infectious causes showed Hydrocephalus.
- 6) Dyslipidemia was a statistically significant risk factor for diabetes, hypertension and CAD cases.
- 7) Patients who regained some neurologic activity after 72 hours were mostly CVA-ischemic stroke .
- 8) Metabolic causes of coma also were partially or fully reversible.
- 9) Low GCS score at admission has a poorer prognosis as similar to AVPU score of “U “ at admission.
- 10) FOUR Score Coma Scale is a very good tool in assessing the short term prognosis of comatose patients, especially after 24 hours when it directly correlates with mortality.

11) FOUR Score Coma Scale is good mainly in comatose patients when in ICU, since it assess brainstem reflexes also.

12) Duration of illness and prognosis has direct correlation, those presenting as acute illness having high mortality within 24 hours of admission.

CONCLUSION

CONCLUSION AND RECOMMENDATIONS

- 1) A detailed history and thorough clinical examination don't have any substitutes.
- 2) Most common cause of non traumatic coma in our study is due to non structural causes
- 3) Age wise,common cause of coma is
 - a) Toxic/metabolic-young age
 - b) Cerebrovascular accident-old age
 - c) Infections-equal in both age groups
- 4) Acute CVA with haemorrhage had the worst prognosis;similar is the cases where there were prolonged cerebral hypoxia like poisoning with respiratory failure.
- 5) Any long duration illness or disease with underlying risk factors also were having poorer prognosis eg:Addisons disease,CKD.
- 6) Rapid progression of already established disease also had bad prognosis eg:CKD uremic encephalopathy,DCLD with hepatic encephalopathy.
- 7) Patients having lower scores at admission had worst prognosis,irrespective of the cause of coma and they had higher 24 hour mortality.
- 8) FOUR score coma scale is the better tool in assessing short term prognosis as it correlates best with 24 hour mortality
- 9) More extensive studies with longer duration and larger population is needed before replacing the prevailing guidelines for coma assessment

10) Higher modalities of investigations like MRI is needed to find out any lesion especially in metabolic causes

11) Long term follow up of patients who gets discharged from the hospital will help us in finding out the various unidentified mechanisms of arousal and coma, especially in cases of metabolic coma.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Bernat J L – Chronic disorders of consciousness Lancet 2006
367:1181-1192.

Diagnosis and treatment of patients who are minimally conscious or vegetative.
2. Booth C M, Boon R H, Tomlinson G et al: is this patient dead, vegetative or severely neurologically impaired. Assessing outcome for comatose survivors of cardiac arrest. JAMA 2004 291: 870 – 879.
3. Stevens R D, Bharadwaj A: Approach to comatose patient. Critical care medicine 2006, 34: 31 – 41 comprehensive review.
4. Monti M M et al : Willful modulation of brain activity in disorders of consciousness NEJM 362: 579; 2010.
5. Posner J B et al: Plum and Posner Diagnosis of Stupor and Coma. 4th edition NewYork Oxford University Press 2007.
6. Wijdicks E F: Disorders of consciousness in Handbook of Clinical Neurology V. 90 3rd series MJ Aminoff et al, Edinburg, Elsevier 2009.
7. Brenner R P2005 the Interpretation of EEG in Stupor and Coma. Neurologist Vol II pg 271 – 284.
8. Wijdicks E F, Cranford R E: 2005, Clinical Diagnosis of Prolonged States of Impaired Consciousness in Adults. Mayoclinic Proc. Vol. 80 pp. 1037 – 1046.

9. Albert M L, Silverberg R, Riches A et al: Cerebral Dominance for Consciousness.
Archi. Neurol. 1976; 453- 454.
10. Joner E G: Cortical and subcortical contributions to activity – dependant plasticity in primate somatosensory cortex.
Annu. Rev. Neurosciences 2000, 23: 1 – 37.
11. Koh M G, Phan T G, Atkinson J L et al: Neuroimaging in deteriorating patient with cerebellar infarcts and mass effects.
Stroke 2000; 31: 2062 – 2067.
12. Laureys S. Science of Society: Death, unconsciousness of brain -
Nat. Rev. Neurosciences 2005; 6: 899 – 909.
13. Minagar A, David N J: Bilateral Infarction in territory of anterior cerebral artery. Neurology 1999; 52: 886 – 888.
14. North J B, Jennett S: Abnormal breathing patterns associated with acute brain damage. Arch. Neurol. 1974; 31: 338.
15. Pareezy J, Damasio A R: Neuroanatomical correlates of brainstem coma
Brain 2003, 126: 1524 – 1536.
16. St. Louis E K, Wijdicks E F, Li – H et al: Predictors of poor outcome in patients with spontaneous cerebellar hematoma.
Can J Neurol. Sci. 2000; 27: 32 – 36.
17. Tesdale G, Jennet B: Assessment of coma and impaired consciousness –
A Practical scale: Lancet 1974; 2: 81 – 84.

18. Young G, Ropper A, and Button C: Coma and impaired consciousness-
A Clinical prospective: NewYork. McGraw Hill 1998: 665.
19. Journal of Neurosurg,psychiatry 2001;71 i13-i17
doi:10.1136/jnp.71.suppl_1.i13
20. Plum F,PosnerJB(1980) the diagnosis of stupor and
Coma(davis,Philadelphia)
21. Beck RW SmithCH(1988)neuroophthalmology(Littlebrown)p242
22. Levy DE,Bates D,Corona JJ etal,(1981)Prognosis in non traumatic coma
Ann Int Med94
23. The Harrisons principles of Internal Medicine 18th edition
24. The Harrisons principles of Internal Medicine 19th ed Mc Graw Hill
25. The Davidsons principles and practice of Medicine 22nd ed Elsevier
26. Bradleys textbook of neurology- 6ed,Elsevier
27. Bikerstaffs neurological examination in Clinical Practice -6 th ed Wiley
28. The Oxford Textbook of Medicine – 5th ed Oxford Univ Press
29. Localisation in clinical neurology 6th edition.Paul W Brazis
30. Cecil Medicine- 24th edition.Elsevier
31. Lukman O F Datti MA Geoffrey O Yussuf AM Musbau R Shakira
OD.Etiology and outcome of medical coma in tertiary care hospital in
Nigeria Ann Nigerian Med 2012-6;92-7
32. Prognosis in patients presenting with non traumatic coma J Emerg Med
2012 mar;42(3)249-53

33. H Adrian Puttgen Romergryko geocadin(2007)Predicting neurological outcome following cardiac arrest J of Neur Scinces 261,108-117
34. Diango D Mogohomaye M Maiga Y Beye SA Dembele AS Coulibaly Y etal Coma in elderly etiology management and prognosis in Dept of Anesthesia and intensive care. Anesthesia 2011b;5:153-7
35. Bates D Caronna JJ cartlidge etal A prospective study of non traumatic coma:Methods and results in 310 patients.Ann Neurol 1977;2:211-20
36. Wong CP Forsyth RJ Kelley TP Eyre J.incidence etiology and outcome of non traumatic coma.a population based study.arch Dis Child 2001;84:193-9
37. Robin S Howard 2008 Coma and Stupor Disorders of consciousness 57-78
38. G Bryan Young (2003)Clinical neurophysiologic assessment of comatose patients Critical care Medicine 31.994
39. Review of medical Physiology-Ganong 24th ed Lange
40. Guyton and Hall textbook of Medical Physiology-12th ed Elsevier
41. Gray's Anatomy-40th ed Churchill Livingstone39

ANNEXURES

PROFORMA

Name

Age

Sex

IP no

Date of admission

Education

Occupation

Address

Informant

Relation with the patient

Duration of illness/Duration since last seen as normal

Precipitating factors/events

Any history of ingestion of poison/chemical/drug overdose

Symptoms preceeding/associated with illness

Past History

Comorbid conditions

Family history

Drug history

Personal history

EXAMINATION

General examination

Consciousness level

Admission	24 hours	48 hours	72hours
-----------	----------	----------	---------

GCS

AVPU

FOUR score coma scale

Vitals

CNS examination:

Higher mental functions

Cranial nerves

Right

Left

1

2

3,4,6

5

7

8

9,10

11

12

Motor system

Bulk

Tone

Power

Reflexes

Any primitive/pathological reflexes

Sensory system

Spinothalamic

Posterior column

Cortical

Cerebellar functions

Gait

Skull and spine

Signs of meningeal irritation/raised ICT

Autonomic nervous system

Other systems

Investigations:

Hb.TC/Dc/ESR/Platelet/PCV/RBC

RBS/Blood Urea/S,Creatinine / S.Electrolytes Sodium, Potassium, Calcium

/LFT/ Lipid profile

TFT/Cardiac biomarkers/Other relevant investigations in selected cases

Urine Complete

ECG in all leads

Chest Xray PA view

CT scan Brain(plain)

MRI Brain with MRA/MRV in selected cases

PATIENT INFORMATION SHEET

Name

Age

Sex

Education

Occupation

Duration of Illnes

Informant

Relation with patient

Education

Occupation

I,-----,-----

----- is willing to include my -----

-- in the study titled EVALUATION OF COMA IN NON HEAD INJURY
CASES

Signature

CONSENT FORM

I,-----,-----

-- have been informed about the above said study in the language known to me
and I give my consent to include ----- in the study

Place

Date

Signature

NEUROIMAGING

NEUROIMAGING

NEUROLEPT MALIGNANT SYNDROME



RIGHT CAPSULOGANGLIONIC ICH WITH IVE



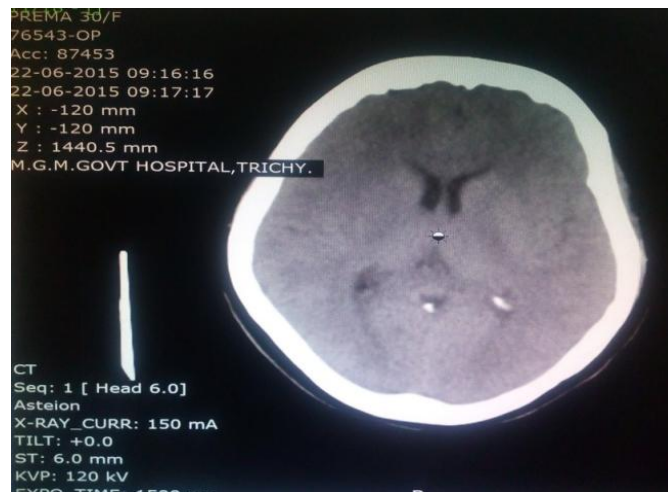
AGE RELATED CEREBRAL ATROPHY



RIGHT MCA INFARCT



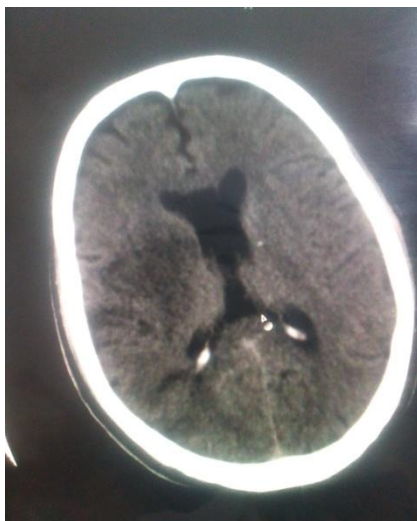
ADDISONIAN CRISIS



UREMIC ENCEPHALOPATHY



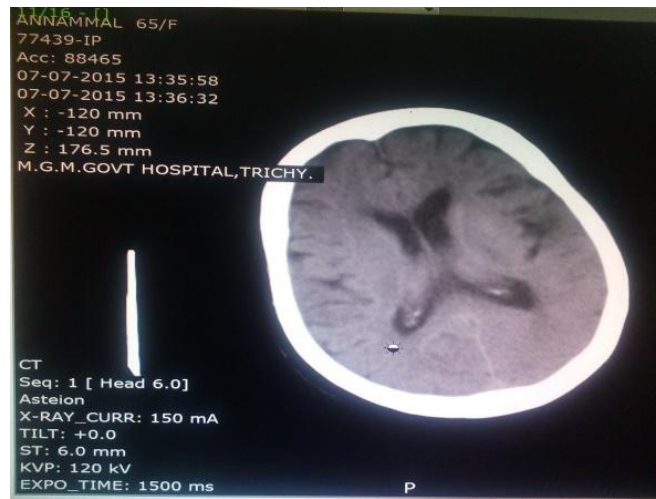
RIGHT BASAL GANGLIA INFARCT



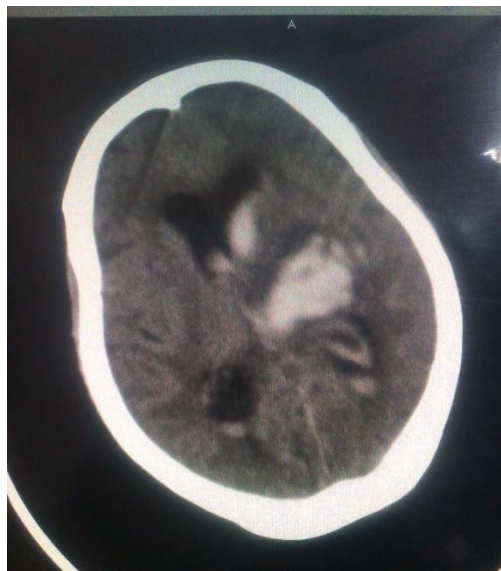
RIGHT TEMPOROPARIETAL ICH WITH EDEMA AND MASS EFFECT



BACTERIAL MENINGITIS



LEFT CAPSULO GANGLIONIC ICH WITH IVE



BILATERAL MCA INFARCT



LEFT TEMPOROPARIETAL ICH WITH IVE WITH MASS EFFECT



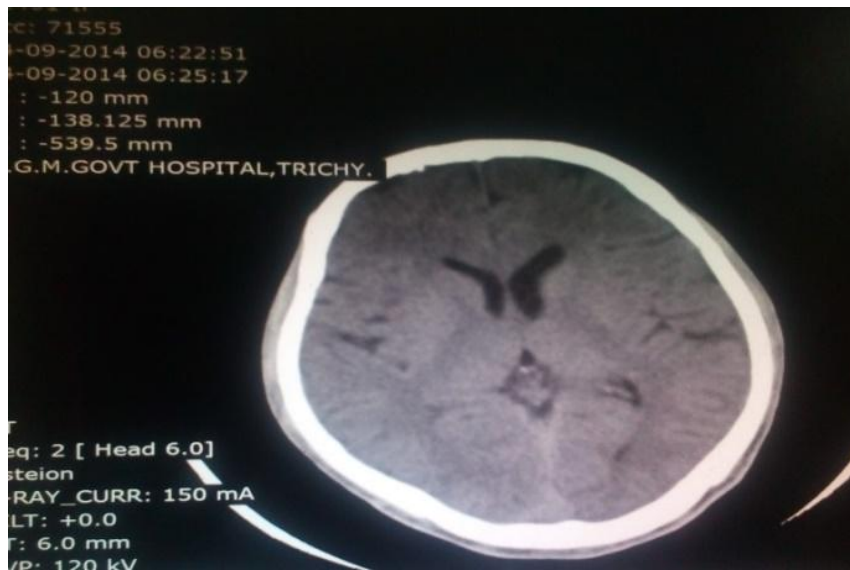
HYDROCEPHALUS



HEPATIC ENCEPHALOPATHY STAGE 4



SNAKE BITE WITH NEUROPARALYSIS



OPC POISONING



MASTER CHART

SL NO	NAME	AGE/SEX	IP NO	D.O.M OF ILLNESS				PRECIPITATING FACTORS	SYMPTOMS PRECEDING/ ASSOCIATED WITH	RISK FACTORS- HTN/DM/CAD/CRO	GCS SCORE AD-2-4HRS	AVPU SCORE AD-2-4HRS	FS CS SCORE AD-2-4HRS	BS	WML	CREATININE	SODIUM	POTASSIUM	CALCIUM	LIPID PROFILE			URE	LET		
				≤9HRS	10-24HRS	25HRS-UP TO 48HRS	≥48HRS													T.CHOL	LDL	HDL		SGOT	SGPT	T.PH
1	maruthamalai	40/F	52828			✓		ANTI PSYCHOTIC INTAKE	FEVER,BODY STIFFNESS	NIL RISK FACTORS	5,8	U P	8 18	104	18	0.9	148	4.1	10.1	187	110	45	NAD	55	56	6.5
2	SETTU	25/M	53306	✓				POISON INTAKE	INCREASED SWEATING, SALIVATION	NIL RISK FACTORS	4,4	U U	6 6	98	31	0.8	135	3.5	9.3	189	120	55	NAD	45	44	6.7
3	MARIYAMMAL	19/F	54356	✓				POISON INTAKE	INCREASED SWEATING, SALIVATION	NIL RISK FACTORS	3,6	U U	6,4	71	90	1	148	3.6	9.5	178	90	46	NAD	38	34	6.5
4	NIRMALA	45/F	58402			✓		ANTI PSYCHOTIC INTAKE	FEVER,BODY STIFFNESS	NIL RISK FACTORS	3,2	U P	5,10	88	27	0.7	144	3.7	8.1	166	78	48	NAD	56	36	7
5	KUMAR	40/M	64166			✓		NIL	NO SYMPTOMS	NIL RISK FACTORS	3,3	U,U	2,2	82	28	0.8	138	3.6	6.5	177	119	56	NAD	44	37	6.5
6	SHSUNDARAM	75/M	6032	✓				NIL	NO SYMPTOMS	HTN,DM,CAD	3,3	U,U	5,5	88	65	1.1	140	4	9.7	210	194	60	1-2 PUS	78	55	7.5
7	MARIYAPPAN	50/M	1196			✓		NIL	TIA	HTN	4,4	U,U	5,5	96	48	1.2	146	3.6	7	190	134	54	NAD	54	36	6.5
8	SALAMMA	70/F	13690	✓				NIL	NO SYMPTOMS	DM	3,14	U,A	8,14	95	64	0.9	136	4	6	221	126	47	NAD	55	37	6.5
9	SANTHA	45/F	13771	✓				NIL	TIA	HTN	5,3	U,U	9,5	120	41	1.1	136	3.8	7	187	115	45	NAD	44	36	6
10	SARAVANAN	18/M	65132	✓				STOPPED INSULIN	NO SYMPTOMS	DM	5,-----	U,-----	7,-----	381	88	1.3	151	4.8	8	221	126	44	PLENITY OF PUS C	223	345	5
11	ANIAMMAL	53/F	59483			✓		NIL	NO SYMPTOMS	HTN	3,3	U,U	2,2	91	48	1.2	148	3.1	7	190	79	49	NAD	78	65	6
12	MUTHUKARUPPAN	23/M	60189	✓				NIL	NO SYMPTOMS	NIL RISK FACTORS	7,5	U,U	6,5	180	28	0.8	136	4.1	8	182	80	56	NAD	67	44	6
13	BALAJI	29/M	19536	✓				NIL	NO SYMPTOMS	NIL RISK FACTORS	3,3	U,U	5,4	145	34	0.9	135	3.8	9	175	87	60	NAD	45	35	7
14	ARULKANI	37/F	8438	✓				SNAKE BITE	UGI BLEED	NIL RISK FACTORS	4,4	U,U	4,2	180	20	1.5	141	3.6	8	178	90	42	3-4 RBC/WPC	33	31	6.8
15	CHITHRA	37/F	8674		✓			STOPPED INSULIN	FEVER	NIL RISK FACTORS	8,12	P,P	12,10	63	46	1	128	7.6	5	184	98	58	SUGAR++++	45	45	6
16	AMIRTHAVALLI	29/F	10376	✓				NIL	NO SYMPTOMS	NIL RISK FACTORS	3,3	U,U	7,3	91	48	0.9	135	3.6	9	167	67	41	NAD	34	21	6.5
17	AMARAVATHY	55/F	10694	✓				SNAKE BITE	BREATHLESSNESS	HTN	4 -----	U,---	3,---	87	38	0.9	143	4	8.7	199	99	49	NAD	43	47	6.7
18	RAM	45/M	61016			✓		NIL	HEMATEMESIS	NIL RISK FACTORS	3,3	U,U	5,1	91	38	1.2	128	4	6	180	87	57	85,BP+	676	512	6
19	IRUDAYARAJ	43/M	17556	✓				NIL	INCREASES SALIVATION	NIL RISK FACTORS	7,7	U,U	6,6	88	48	1.1	148	4.6		187	123	35	NAD	56	42	7
20	ARIYAN	70/M	15436			✓		NIL	FEVER	NIL RISK FACTORS	3,...	U,...	2 -----	126	76	1.3	126	4.1	8.6	184	100	49	NAD	35	62	6
21	SATHYAPRIYA	19/F	15380			✓		NIL	FEVER HEADACHE	NIL RISK FACTORS	5,8	U,U	5,1	122	24	0.8	133	3.7	7.5	190	100	47	NAD	41	34	6
22	RAJENDRAN	50/M	15206			✓		NIL	FEVER	NIL RISK FACTORS	5,5	U,U	7,6	114	45	0.7	130	3.6	6	165	85	46	NAD	45	38	6.3
23	MALLIKA	42/F	16195			✓		NIL	FEVER,DYSURIA	DM	10,8	P,P	11,10	108	109	9.8	131	5.3	5	196	167	43	PLENITY OF PUS CELLS	90	106	6.6
24	PERIYAKKAL	40/F	19048	✓				POISON INTAKE	BREATHLESSNESS	NIL RISK FACTORS	3,...	U,...	3,---	141	45	0.9	140	3.9	7	187	89	42	3-5 PUS CELLS	48	36	6.5
25	YAGATHAKJ	62/M	2149	✓				NIL	TIA	HTN	4,...	U,...	7,---	112	55	1.6	136	4	7.6	224	90	45	NAD	58	31	5
26	VINOTHKUMAR	24/M	21775			✓		NIL	FEVER,HIGH COLORED URINE	NIL RISK FACTORS	3,...	U,---	1,---	148	63	1.4	146	4.8	7	182	98	57	85,BP+	518	424	7
27	INDRANI	58/M	21994			✓		NIL	VOMITING	NIL RISK FACTORS	7,6	P,P	12,11	128	38	1.2	136	4.2	6.4	180	96	54	NAD	58	49	6
28	SAITHUN	70/F	21136			✓		NIL	TIA	HTN,DM,CAD	9,9	P,P	13,13	122	28	1.1	136	4.8	5.9	198	156	45	NAD	48	31	9

ECG	CXR-PA	CT BRAIN	DIAGNOSIS	OTHER INV	MORTALITY WITHIN 24 HRS		
						24-48 HRS	48-72 H
NSR	WNL	NORMAL	NEUROLEPT MALIGNANT SYNDROME	S.CPK=145	NO	NO	NO
S TACHYCARDIA	WNL	NORMAL	OPC POISONING WITH RESP.FAILURE		YES		
S TACHYCARDIA	WNL	NORMAL	OPC POISONING WITH RESP.FAILURE		YES		
NSR	WNL	NORMAL	NEUROLEPT MALIGNANT SYNDROME	S.CPK=48	NO	NO	NO
NSR	WNL	MALIGNANT INFARCT LEFT MCA WITH MASS EFFECT	CVA R HEMIPLEGIA- ISCHEMIC STROKE		NO	NO	NO
NSR	WNL	RIGHT FRONTO TEMPORAL ICH WITH IVE;SAH	CVA L HEMIPLEGIA- HEMORRHAGIC STROKE		NO	NO	YES
NSR	WNL	LEFT CAPSULO GANGLIONIC ICH	CVA L HEMIPARESIS		NO	YES	
NSR	WNL	NORMAL	HYPOLYCEMIA		NO	NO	NO
NSR	WNL	BRAINSTEM ICH	CVA-HEMORRHAGIC STROKE		NO	NO	YES
NSR	WNL	NORMAL	SEPTIC SHOCK/UROSEPSIS	U C&S: CANDIDA	YES		
NSR	WNL	RIGHT CAPSULOGANGLIONIC ICH WITH IVE	CVA L HEMIPLEGIA- HEMORRHAGIC STROKE		YES		
NSR	WNL	NORMAL	KURUNAI POISONING WITH RESP FAILURE		YES		
NSR	WNL	NORMAL	ATTEMPTED HANGING WITH HIE		YES		
NSR	WNL	BRAINSTEM ICH	SNAKE BITE WITH COAGULOPATHY	WBCT >20 MIN	NO		
NSR	WNL	NORMAL	HYPERGLYCEMIC HYPEROSMOLAR STATE		NO	NO	YES
NSR	WNL	NORMAL	ATTEMPTED HANGING WITH HIE		NO	YES	
NSR	WNL	NORMAL	SNAKE BITE WITH NEUROPARALYSIS		YES		
NSR	WNL	NORMAL	DCLD HEPATIC ENCEPHALOPATHY STAGE 4	S.BR=12.1(T)	NO	YES	
S TACHYCARDIA	WNL	NORMAL	OPC POISONING WITH RESP.FAILURE		NO	NO	NO
NSR	WNL	NORMAL	GBS WITH RESP.FAILURE	CSF:PROTEIN INC.	YES		
NSR	WNL	DIFFUSE CEREBRAL EDEMA	MENINGOENCEPHALITIS?TBM	CSF:PR-1.6 SUG 52 10 CELL	NO	YES	
NSR	R U.LOBE FIBROSIS	DIFFUSE CEREBRAL EDEMA	TBM	CSF:PR 2G	NO	YES	
S TACHYCARDIA	WNL	NORMAL	T2DM/UROSEPSIS	U C&S CANDIDA	NO	NO	NO
NSR	WNL	NORMAL	LYZOL POISONING, RESP FAILURE		NO	YES	
NSR	CARDIOMEGALY	BILATERAL CEREBELLAR ICH WITH BRAINSTEM ICH WITH IVE	CVA-HEMORRHAGIC STROKE		NO	YES	
NSR	WNL	NORMAL	HEPATIC ENCEPHALOPATHY ST 4		NO	YES	
NSR	WNL	NORMAL	DCLD HEPATIC ENCEPHALOPATHY STAGE 4	INR 1	NO	NO	NO
NSR	CARDIOMEGALY	MASSIVE LEFT MCA INFARCT	CVA RT HEMIPLEGIA- ISCHEMIC STROKE		NO	NO	NO

29	RAMU	45/M	21068			✓		NIL	UGI BLEED	NIL RISK FACTORS	3...	U...	3...	78	55	1	135	3.6	9	178	87	49	NAD	116	82	5
30	MURUGESAN	48/M	18922	✓				NIL	UGI BLEED	NIL RISK FACTORS	6.6	P,P	12.9	115	31	0.8	140	4.6	8	188	84	65	NAD	70	51	6
31	GURUNATHAN	60/M	19490			✓		NIL	JAUNDICE	NIL RISK FACTORS	3...	U...	2...	96	49	1.2	137	4	8.6	170	80	48	NAD	81	86	5.5
32	PATTANI	57/M	19568			✓		NIL	FEVER HEADACHE	HTN	4...	U...	6...	116	48	1.1	144	3.6	9	182	89	48	NAD	71	36	6
33	BALAKRISHNAN	79/M	19760		✓			NIL	NO SYMPTOMS	NIL RISK FACTORS	3...	U...	5...	106	33	0.7	133	3.4	7.6	171	70	39	NAD	41	40	6
34	SELVAM	45/M	19673	✓				SNAKE BITE	BREATHLESSNESS	NIL RISK FACTORS	3...	U...	7...	124	16	0.7	140	3.8	7.9	180	83	44	NAD	48	41	6.8
35	SUDATHA	19/F	20027			✓		NIL	BREATHLESSNESS	NIL RISK FACTORS	11.11	P,P	15.15	110	28	1.6	144	4	7	187	81	48	NAD	108	96	6.9
36	SEKAR	46/M	20011	✓				NIL	TIA	HTN	6.4	U,U	12.6	142	38	1.3	155	4	6	218	99	47	NAD	40	35	7
37	MUTHYAN	45/M	18969	✓				NIL	NO SYMPTOMS	DM	9.7	P,P	14.14	100	48	0.7	136	4	8	200	177	45	NAD	40	35	6.3
38	PANNEERSELVAM	60/M	20514	✓				NIL	NO SYMPTOMS	HTN	6.6	P,P	13.13	144	70	1.6	148	3.3	7.3	245	91	55	NAD	46	41	8.1
39	MARUDANAYAGAM	65/M	20776	✓				NIL	NO SYMPTOMS	HTN	9.8	U,U	13.12	128	41	0.9	137	3.6	8	199	90	55	NAD	50	48	8
40	PITCHAIPELAI	60/M	22772	✓				NIL	NO SYMPTOMS	DM	6.8	U,U	8.10	98	44	1.6	138	4.5	9	213	164	51	NAD	60	37	7
41	AROKIYADASS	43/M	21414			✓		NIL	NO SYMPTOMS	HTN	8.8	U,U	13.13	102	38	0.9	140	4.1	8	211	91	61	NAD	66	45	8
42	LAKSHMI	82/F	21681	✓				NIL	NO SYMPTOMS	HTN,DM,CAD	6.6	U,U	13.13	78	90	1.1	128	4	7.6	245	145	47	3-SPUS CELLS	70	60	6
43	POJAN	40/M	24477			✓		NIL	NO SYMPTOMS	HTN	3...	U...	5...	102	48	1.6	131	3.8	8.8	180	100	49	NAD	55	34	7
44	SANTHRI	50/F	24482			✓		NIL	NO SYMPTOMS	HTN,CKD	9.9	P,P	13.11	108	249	14.8	128	5.8	7	179	109	55	NAD	67	39	6.1
45	SUBRAMANI	58/M	25687	✓				ATTEMPTED HANGING	NO SYMPTOMS	NIL RISK FACTORS	6.6	U,U	8.9	166	45	0.9	134	3.8	8	185	100	67	NAD	48	37	6.6
46	RAVI	47/M	25712	✓				NIL	BREATHLESSNESS	NIL RISK FACTORS	3...	U...	5...	91	48	1	127	3.6	7	188	101	44	NAD	70	56	6
47	NAJMUNUSHA	22/F	725	✓				NIL	NO SYMPTOMS	NIL RISK FACTORS	9...	U...	13...	148	21	0.7	136	4.2	6	190	100	45	NAD	48	31	7
48	DHANAM	60/F	1697	✓				SNAKE BITE	BREATHLESSNESS	NIL RISK FACTORS	8.8	U,U	9.9	116	82	2.6	134	4.9	8	189	96	47	NAD	49	32	8
49	THIRUPATHI	42/M	3695	✓				NIL	NO SYMPTOMS	NIL RISK FACTORS	4.3	U,U	10.8	88	47	0.9	138	4.6	7.8	194	89	44	NAD	48	36	6.7
50	VIAAYAKUMAR	27/M	4709	✓				ATTEMPTED HANGING	NO SYMPTOMS	NIL RISK FACTORS	6.8	U,U	8.10	80	22	0.7	139	4	6	178	89	46	NAD	55	32	6.5
51	MARUTHAMBAL	65/F	6053			✓		NIL	FEVER HEADACHE	DM	8.3	P,U	13.2	92	90	1.3	140	4.1	8	199	176	55	NAD	45	39	6.6
52	SAPPANI	70/M	3286			✓		NIL	BREATHLESSNESS	CKD	4...	U,U	6...	98	123	10.1	128	5.9	9	167	82	57	NAD	45	23	5.6
53	SELVAKUMAR	30/M	9730	✓				NIL	FEVER HEADACHE	NIL RISK FACTORS	6...	U...	8...	87	31	0.9	134	3.6	6	188	93	53	NAD	34	27	6.7
54	LAKSHMI PELAI	54/M	12377		✓			SNAKE BITE	NO SYMPTOMS	NIL RISK FACTORS	8.4	U,U	9.9	99	78	4.5	132	5.5	8	184	85	40	NAD	55	34	6.4
55	THANGAM	22/M	12607	✓				SNAKE BITE	NO SYMPTOMS	NIL RISK FACTORS	10.8	P,U	13.10	83	67	5.5	129	4.3	7	182	98	40	NAD	34	44	6.1
56	GAYATHRIDEVI	21/F	14103	✓				POISON INTAKE	NO SYMPTOMS	NIL RISK FACTORS	3...	U...	7...	123	31	0.7	136	4.7	8	190	85	56	NAD	654	1787	6
57	IRUDAYARAJ	74/M	16491		✓			NIL	TIA	HTN	11.10	P,P	14.12	87	33	0.9	137	4.5	7.7	198	98	47	NAD	55	41	5
58	PREMA	58/F	4484	✓				ADDISON'S DISEASE	BREATHLESSNESS	NIL RISK FACTORS	8...	U...	10...	79	31	1	156	5.7	9	189	92	67	NAD	44	29	5.6
59	THIRUPATHI	65/M	4394		✓			NIL	TIA	HTN	7.5	U,U	9.6	95	42	1.1	144	4.9	7.9	210	95	45	NAD	33	23	5.9
60	ANNAMMAL	67/F	4102			✓		NIL	FEVER HEADACHE	NIL RISK FACTORS	6.3	U,U	8.5	105	37	0.6	131	4.2	8.5	204	108	56	NAD	45	37	6

NSR	R.PLEURAL EFFUSION	NORMAL	DCLD/UGI BLEED/ HE STAGE4		YES		
S BRADYCARDIA	BILATERAL PULMONARY CONGESTION	NORMAL	OPC POISONING WITH RESP.FAILURE		NO	N	NO
NSR	WNL	NORMAL	HEPATIC ENCEPHALOPATHY ST 4	S.BR=9.4 (T)	YES		
NSR	WNL	HYDROCEPHALUS	BACTERIAL MENINGITIS		YES		
NSR	WNL	NORMAL	UNKNOWN TABLET POISONING WITH RESP.FAILURE		YES		
NSR	WNL	AGE RELATED CEREBRAL ATROPHY	SNAKE BITE WITH NEUROPARALYSIS AND COAGULOPATHY		YES		
NSR	R LOWER LOBE CONSOLIDATION, COLLAPSE	HYDROCEPHALUS			NO	NO	NO
LVH	WNL	BRAINSTEM ICH	SEPTIC SHOCK/MODS		NO	NO	YES
NSR	WNL	MASSIVE RIGHT MCA INFARCT	CVA HEMORRHAGIC STROKE		NO	NO	NO
LVH	WNL	RIGHT CAPSULOGANGLIONIC ICH	CVA L HEMIPLEGIA- ISCHEMIC STROKE		NO	NO	NO
LVH	WNL	BRAINSTEM ICH	CVA LHEMIPLEGIA- HEMORRHAGIC STROKE		NO	NO	NO
NSR	WNL	AGE RELATED CEREBRAL ATROPHY	CVA-HEMORRHAGIC STROKE		NO	NO	NO
LVH	WNL	ACUTE L MCA INFARCT, OLD R MCA INFARCT	ATTEMPTED HANGING WITH HIE STAGE 3		NO	NO	NO
LVH	CARDIOMEGALY	BRAINSTEM ICH	RECURRENT CVA R HEMIPARESIS		NO	NO	NO
NSR	WNL	BRAINSTEM ICH	CVA HEMORRHAGIC STROKE		NO	NO	NO
LVH WITH STRAIN	CARDIOMEGALY	BRAINSTEM ICH	CVA HEMORRHAGIC STROKE		YES		
NSR	WNL	NORMAL	UREMIC ENCEPHALOPATHY		NO	NO	NO
NSR	FIBROTIC STRANDS BOTH LUNGS	NORMAL	ATTEMPTED HANGING WITH HIE STAGE 3		NO	NO	NO
NSR	WNL	RIGHT TEMPOROPARIETAL ICH WITH IVE, HYDROCEPHALUS	MARQUIS DISEASE/ RESTRICTIVE LUNG DS/RESP FAILURE		YES		
NSR	WNL	NORMAL	CVA L HEMIPLEGIA- HEMORRHAGIC STROKE		YES		
NSR	WNL	MASSIVE LEFT MCA INFARCT	SNAKE BITE WITH NEUROPARALYSIS		NO	NO	NO
NSR	WNL	NORMAL	CVA R HEMIPLEGIA- ISCHEMIC STROKE		NO	YES	
NSR	WNL	AGE RELATED CEREBRAL ATROPHY, HYDROCEPHALUS	ATTEMPTED HANGING WITH HIE		NO	NO	NO
NSR	WNL	NORMAL	MENINGOENCEPHALITIS- BACTERIAL		YES		
NSR	WNL	HYDROCEPHALUS	CKD UREMIC ENCEPHALOPATHY		NO	NO	YES
NSR	WNL	NORMAL	BACTERIAL MENINGITIS		NO	YES	
NSR	WNL	NORMAL	SNAKE BITE, UREMIC ENCEPHALOPATHY		NO	YES	
NSR	WNL	NORMAL	SNAKE BITE, UREMIC ENCEPHALOPATHY		NO	YES	
NSR	WNL	NORMAL	RAT KILLER PASTE POISONING, METABOLIC ENCEPHALOPATHY		NO	NO	YES
NSR	WNL	BRAINSTEM ICH	CVA HEMORRHAGIC STROKE		NO	NO	NO
S TACHYCARDIA	EMPHYSEMATOUS CHANGES	NORMAL	ADDISONIAN CRISIS		YES		
S BRADYCARDIA	WNL	MASSIVE LEFT MCA INFARCT	CVA R HEMIPLEGIA- ISCHEMIC STROKE		NO	YES	
NSR	WNL	HYDROCEPHALUS	BACTERIAL MENINGITIS		NO	YES	

KEY TO MASTER CHART

(L) MCA-LEFT MIDDLE CEREBRAL ARTERY

(R) MCA-RIGHT MIDDLE CEREBRAL ARTERY

AD –ADMISSION

AVPU-ALERT, VOICE, PAIN, UNCONSCIOUS

BS/BP- BILE SALTS, BILE PIGMENTS

CAD-CORONARY ARTERY DISEASE

CKD-CHRONIC KIDNEY DISEASE

CSF-CEREBRO SPINAL FLUID

CVA-CEREBROVASCULAR ACCIDENT

DCLD-DECOMPENSATED LIVER DISEASE

DM-DIABETES MELLITUS

FSCS-FOUR SCORE COMA SCALE

GCS-GLASGOW COMA SCALE

HDL-HIGH DENSITY LIPOPROTEIN

HIE-HYPOXIC ISCHEMIC ENCEPHALOPATHY

HTN-SYSTEMIC HYPERTENSION

ICH-INTRA VENTRICULAR HEMORRHAGE

IVE-INTRAVENTRICULAR EXTENSION

LDL-LOW DENSITY LIPOPROTEIN

LFT-LIVER FUNCTION TEST

LVH-LEFT VENTRICULAR HYPERTROPHY

NAD-NO ABNORMALITIES DETECTED

NSR-NORMAL SINUS RHYTHM

OPC-ORGANO PHOSPHOROUS COMPOUND

R L LOBE-RIGHT LOWER LOBE

R U LOBE-RIGHT UPPER LOBE

RBS-RANDOM BLOOD SUGAR

S.BRADYCARDIA—SINUS BRADYCARDIA

S.BR-SERUM BILIRUBIN

S.CPK-SERUM CREATININE PHOSPHOKINASE

S.TACHYCARDIA-SINUS TACHYCARDIA

SAH-SUB ARACHNOID HEMORRHAGE

T.PR-TOTAL PROTEIN

TBM-TUBERCULOUS MENINGITIS

TIA-TRANSIENT ISCHEMIC ATTACKS

UGI-UPPER GASTRI INTESTINAL

URE-URINE ROUTINE EXAMINATION

URINE C& S-URINE CULTURE AND SENSITIVITY

WBCT-WHOLE BLOOD CLOTTING TIME

WNL-WITHIN NORMAL LIMITS



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In partial fulfillment of the regulations
For the award of the degree of
M.D. General Medicine – [Branch: I]
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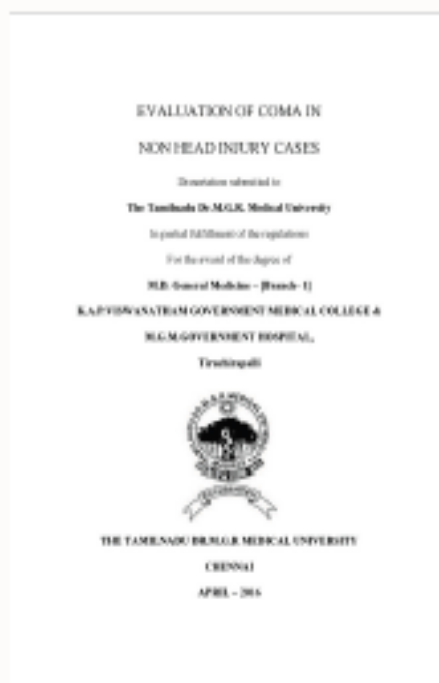


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ABBREVIATIONS

ADEM-ACUTE DEMYELINATING ENCEPHALOMYELITIS

AKI-ACUTE KIDNEY INJURY

AVPU-ALERT,VOICE,PAIN,UNCONSCIOUSNESS

CJD-CRUZEFELT JAKOB DISEASE

CKD-CHRONIC KIDNEY DISEASE

CSF-CEREBRO SPINAL FLUID

CT-COMPUTED TOMOGRAPHY

CVA-CEREBROVASCULAR ACCIDENT

DCLD-DECOMPENSATED LIVER DISEASE

ECG-ELECTRO CARDIOGRAM

EEG-ELECTRO ENCEPHALOGRAM

GBS-GULLIAN BARRE SYNDROME

GCS-GLASGOW COMA SCALE

HIE-HYPOXIC ISCHEMIC ENCEPHALOPATHY

ICH-INTRACEREBRAL HEMORRHAGE

ICT-INTRA CRANIAL TENSION

IVE-INTRAVENTRICULAR EXTENSION

MCA-MIDDLE CEREBRAL ARTERY

MODS-MULTIORGAN DYSFUNCTION SYNDROME

MRI-MAGNETIC RESONANCE IMAGING

OPC-ORGANOPHOSPHOROUS COMPOUND

RBS-RANDOM BLOOD SUGAR

SAH-SUB ARACHNOID HEMORRHAGE

TBM-TUBERCULOUS MENINGITIS